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Original Articles.

ON THE SIMULATION OF HYSTERIA BY ORGANIC DISEASE OF THE NERVOUS SYSTEM.[1]

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In our practice as physicians we are continually meeting with cases which suggest the presence of organic lesion of the nervous system, whilst, at the same time, circumstances occur to make us doubt whether the symptoms present may not be dependent upon what, for want of a better term, is styled functional disorder. It is quite unnecessary here to attempt to define what is meant by the term, nor to insist upon the importance, both as regards prognosis and the treatment of the condition, that our diagnosis should be correct.

The field occupied by cases of this description is so vast that it would be ridiculous for me to attempt anything like a survey of it. I propose therefore on this occasion to limit my observations to those instances in which some loss of power in a limb or limbs is the dominating feature. The admirable paper which was recently read before this Society by Dr. Hughes Bennett on "Muscular Hypertonicity" will be very present to your minds as discussing those numerous examples which wear the semblance of spastic paraplegia, and with these therefore it will be unnecessary for me to deal. Nor

[1] Presidential Address at the Annual Meeting of the Neurological Society of London on January 23rd, 1890.

shall I dwell much upon the more common forms of so-called hysterical paralysis, the recognition of which the surrounding circumstances may render very easy. In fact I cannot hope to do more than discuss a few points of interest and difficulty, without pretending to any attempt at exhausting an exceedingly wide subject.

One difficulty in connection with the class of cases to which I refer lies in the fact that under the term functional we probably have often to include (because of the impossibility of differentiating them) examples of various forms. There is, in the first place, involuntary simulation of organic disease associated with some morbid psychical condition to which the term hysterical is usually applied. Next, there are examples of functional inability dependent probably upon a temporary malnutrition of certain parts of the nervous centres. There appears reason to believe that this local imperfection may sometimes exist for a long while without giving rise to destructive changes in the parenchyma, but that in many instances it represents but a stage of actual change in tissue which eventually becomes permanent and irrecoverable. How far we may reckon that there is a point in this progressive deterioration at which treatment may be successfully applied must still be considered doubtful, although evidence tends rather to the idea that this is probably the case. The cases of this class present peculiar difficulty in their diagnosis, because, combined with evidence of local troubles of nutrition in the nerve centres, there are often so many symptoms of hysterical or emotional character as to greatly obscure and confuse the picture.

It is especially to this category that I propose to draw attention on the present occasion. I fear that my remarks will necessarily tend rather to expose than to solve the difficulties which we are liable to encounter in dealing with this question, and that their discursive character may suggest, what is indeed the truth, that I am rather employed in thinking aloud than in imparting information. As far as possible I shall relate the sequel of cases to which reference is made. This will often decide a question which has been a puzzling one. Owing to the kindness of my friend, Dr.

Playfair, with whom I have examined during the last ten years many cases in which a serious question of diagnosis has arisen, I shall be able to refer to the results of the Weir Mitchell treatment applied with his well-known skill. The physician truly experiments, as Claude Bernard has said, when he tries to bring about a modification in the symptoms of disease. In some cases, to which reference will be made, the disappearance under improved nutrition of the symptoms which gave rise to the doubt has resolved the difficulty in the happiest way possible. In others the question has been determined by failure to interrupt the course of the malady, or by a fatal issue.

There is a form of paraplegia of which I have seen several examples, in nearly all of which important questions have arisen as to the functional or organic nature of the condition. The subject will be best introduced by briefly relating particulars of a few cases.

A young lady, æt. twenty-one, was seen by me in consultation with Dr. Playfair on November 1st, 1886.

Since the age of fifteen she had been observed by her friends to have a peculiar gait, and this was noticed two years later by a surgeon who was consulted on account of some pain in her joints. At that time she walked with a stiff and stilted gait, which was chiefly noticeable in going upstairs, and varied very much. There appeared, however, I was told, to be no want of muscular power. She could walk well, and dance. From time to time she suffered from a variety of typical hysterical symptoms, including an aversion from food, under which she had greatly lost flesh and strength at the time that I saw her. It was especially during several preceding months that she had gradually failed in her walking powers, and on several occasions had fallen down.

The question to be determined was whether the condition was an hysterical one, as it had been pronounced to be by more than one medical authority, and likely therefore to yield to a course of Weir Mitchell treatment, for which purpose she had been taken to Dr. Playfair.

On examination I observed that the girl was highly anæmic. She could stand or walk without support, but

slowly and laboriously, inclining the trunk to the side opposite to that from which depended the limb which was being advanced. When in the recumbent position she could flex the thighs upon the trunk with a fair amount of force, but when seated she had little or no power of lifting either knee. Extension at the knee joint was only moderately well performed, whilst the power of flexion at the same joint, and extension and flexion at the ankle joint, appeared to be normal. When standing it was with great difficulty that she could place her right foot upon the sofa, and only by adopting a swinging movement. She could not even swing the left foot on to it. With her right foot on the sofa she could not lift herself up so as to stand upon it. The muscles of the lower extremities were well developed. There were no sensory symptoms. No complaint was made of the upper extremities.

The wrist jerks were exaggerated; the knee jerks, on the other hand, were decidedly feeble. This contrast in the state of the deep reflexes, when taken in conjunction with the other symptoms, gave important evidence. I have often referred to the fact that the knee jerk is not, according to my observation, lost in cases of hysteria; on the contrary, it is almost always increased. Its marked diminution in this case, alongside of greatly exaggerated wrist jerk, pointed in the circumstances to impaired nutrition of the second lumbar segment of the spinal cord. The complete loss of power in the iliaco-psoas muscles, as shown by the examination of the patient's movements, indicated that the first lumbar segment was still more severely affected. An opinion to this effect was given. As regards the Weir Mitchell treatment, it was agreed that the general health was likely to receive benefit from it, but that no promise could be made that the local symptoms would be removed.

The patient went through a course of systematic treatment conducted by Dr. Playfair, who at the end of six weeks was kind enough to let me again examine her. She was no longer anæmic, and had greatly improved in appearance. She had gained fifteen pounds in weight. The knee jerks were now well marked, but there was still considerable

difficulty in the movements performed by the iliaco-psoas muscles. She could "fling" her foot on to a low chair—it could scarcely be called lifting it—and could then drag herself up, holding on to the back of the chair, so as to stand upon the seat. This was only when the right foot was employed.

I am informed that this lady still shows a somewhat peculiar gait, but that she can dance, run about quite actively, ascend stairs quickly, and appears to have full muscular power. She was recently married.

Some four years previous to this I had been consulted in the case of a well-nourished healthy-looking young woman, æt. twenty-four, who had failed in her walking powers, and a similar question had arisen as regards the nature of her disability. It is notorious that when a girl of highly neurotic temperament complains of difficulty in walking, the suggestion of an hysterical cause is very apt, and in many cases justly, to arise.

The patient, whom I saw in 1882, had been observed by her mother in 1879 to be walking in a laboured way, which was at first attributed to the wearing of high-heeled boots, and the presence of chilblains. But the difficulty continued and only increased as time went on. It appeared that in the winter of 1876-77 she had undergone great fatigue in "rinking," and it was not long after this that she had begun to feel unduly tired in walking.

I found on examination that the muscles of the lower extremities were well developed, and that the only defect in movement was in the action of the iliaco-psoas group. There appeared to be absolutely no power of flexing the thigh upon the pelvis, all the other movements being well performed. The knee jerks were normal. The patient was sent to bed, fed, rubbed, and fired in the lower dorsal region of the spine, but without any effect. In December 1883 she could still walk, but required first to be set upon her feet.

I learned last year that she had lost all power of movement in her lower extremities. There was no wasting of the limbs, which were sound and indeed unduly firm.

Last year a sister of this patient, æt. twenty-eight, was

brought to me suffering in her turn from a somewhat narrowly localised loss of power. This patient could run up stairs two steps at a time, and mount easily upon a chair without the aid of her arms, but she had no power of dorsal flexion of the right foot, and almost none in the left. The leg itself was very firm and plump. Electrical examination shewed complete absence of reaction to induced currents in the anterior tibial muscles.

The patient, I learned, had enjoyed very good health, but her walk had been observed to be peculiar for eight or nine months. This was due, no doubt, to the dropping of her feet necessitating high action. Besides the patient with iliaco-psoas paralysis above described, this girl has two other sisters, one whom I have not seen, who is thirty-two years old and married, and is described as walking in the same fashion. She is unable to walk fast. The other sister, twenty-five years of age, shows no peculiarity of gait. It is evident that in the first of these two sisters' cases no question as between the organic or functional nature of the affection could have arisen, had the muscles affected (the flexors of the thigh) been in a position to be explored by electric currents as they were in the second. But although in very emaciated persons it may, though very rarely, be possible to apply a rheophore in the situation of the iliacus muscle, the pain caused by electric currents in this situation is intolerable to most persons, and renders electrical exploration impracticable.

I suppose it can hardly be doubted that the condition of the anterior tibial muscles, in the second sister, gives the clue to that of the iliaco-psoas muscles in the first, and from the description given of the married sister (whom I have not seen) it seems likely that she is also affected in some of her muscles in a similar manner. It seems probable that these are cases of simple idiopathic muscular atrophy which is apt to occur in several members of the same family, is often inherited, and is not of central origin. The remarkably firm condition of the limbs, coupled with the results of electrical examination, seems to point to overgrowth of interstitial tissue. In this family I could hear of no other instance of a like affection, and the only peculiarity in the history that

I could learn was that the father and mother were first cousins.

Some years ago I saw with Dr. Playfair another lady who had been affected for more than ten years with a similar powerlessness confined to the iliaco-psoas muscles. In her case the Weir Mitchell treatment was adopted without success. She was probably an example of the same condition.

In 1885, a military man, æt. sixty-eight, consulted me on account of inability to flex the hip joints—the other movements of the lower extremities being perfect, and the electrical reactions normal. There was no loss of sensation. The knee jerks were perfect, and there was no difficulty in the action of the bladder. The want of power first showed itself at the close of the Crimean campaign, during which he had enjoyed good health. He was affected with peculiar nervous sensations. In this case there was a history of syphilis. Specific treatment had produced no effect.

Atrophy of the iliaco-psoas muscles occurring in a young female, accompanied by hysterical symptoms, would seem to be peculiarly liable to be wrongly interpreted. The patient complains of difficulty in walking, but the knee jerks are preserved, and the electrical condition of all the muscles of the lower extremity that can be tested is strictly normal. One is naturally disposed to expect in such a case that if there be a central lesion in the cord, either the muscles innervated from the lumbar enlargement will be found atrophied and their electrical reactions abnormal, or if the lesion be above the enlargement, symptoms of spasticity will be present, the knee jerks will be exaggerated, and there will be more or less ankle clonus.

I think that the cases which I have described may serve a useful purpose in drawing attention, in a doubtful case, to the fact that there may be isolated atrophy of the iliaco-psoas.

The successful result of systematic treatment by high feeding and massage in the case first related is probably to be explained by the fact that the patient was anæmic and much reduced. It seems likely that in her case the condi-

tion was not one of congenital idiopathic muscular atrophy, but that there was a temporary state of mal-nutrition of the cord, which recovered. I remember another instance of the kind in a young woman profoundly affected with anæmia, who regained power under appropriate treatment.

A governess, æt. twenty-three, came to me on Oct. 18th, 1884, on account of difficulty in walking. She had been delicate in childhood, but had usually enjoyed good health. For the last fortnight she had been unable to carry on her occupation. Five weeks previously she had felt her breath very short in going up stairs, and had kept in bed for a few days. Every time she left the bed she fainted. A fortnight since she experienced great difficulty in walking, as though her legs gave her no support, and there was a great aching at the bottom of the back. She could only just hobble about from one room to the other. There was great vesical irritability. The catamenia had been suspended for three months. The patient was suffering from marked chloro-anæmia; the cardiac sounds were normal; the urine contained no albumen nor sugar. There was some tenderness on pressure on the first lumbar vertebra; the spinal column showed no deformity. The movements of the lower extremities were feeble, especially flexion of the thigh upon the hip, which could scarcely be performed. The knee jerks were rather in excess. Iron and arsenic were prescribed for her.

After a fortnight she had greatly improved in colour and was able to walk better. The vesical trouble, however, still continued. She was unable to lift the left knee, but could lift the right. There was no affection of sensibility. In three weeks more she felt quite well in herself and the vesical trouble had ceased. She was able to lift the right knee against a certain amount of pressure, but not the left at all. She could straighten the left leg, flex and extend the foot, and in fact do anything but lift the knee. In going up stairs she would put her right foot down first, and then draw the left after her; in descending the left was put down first. She still suffered from palpitation and breathlessness.

About six weeks later, when I saw her again, the legs

had quite recovered; she was able to walk more than a couple of miles, and could lift her knee on either side in normal fashion.

It will be remembered that the late Dr. Moxon, in his Croonian Lectures before the Royal College of Physicians in 1881, called attention to the great length and obliquity of the spinal arteries which supply the lower third of the spinal cord, as a probable cause of temporary anæmia. It would seem that where there is general anæmia this anatomical arrangement may explain the localisation of disturbed nutrition in this part. I suppose that in the case first related, where the knee jerks, which were at first small, became well pronounced as the patient recovered, it was to the anæmic condition of the cord at the second lumbar segment that the imperfection of the reflex action was due. One can conceive it possible that anæmia in a given case might be so extreme as to cause a temporary disappearance of the reflex. It is necessary to bear this in mind, as the absence of knee jerk is a symptom which, if the illness has been long continued, is more suggestive of irreparable damage than of a condition capable of being treated successfully.

The absence of knee jerk, so far as my experience goes, in a young female is a matter requiring very careful examination. I need not mention the obvious conditions which usually explain the absence, but would refer to cases of a less common kind, which it is well to bear in mind. One of these I saw with Dr. Playfair in 1884. The patient, a young lady, aged twenty-six, had begun to get weak in walking eight or nine years previously. The difficulty had gradually increased until she had an attack of typhoid fever, since when she had been unable to walk at all.

Examination showed the following condition:—Her speech is ataxic, the words being slurred. The hands possess a good grasp, but are rather wildly moved about when directed to touch an object—a condition of marked ataxy. This is the case with each hand. It is not the tremor of disseminated sclerosis, but the movement of a person suffering from tabes dorsalis. There is no wrist jerk on either side. She cannot sit up. When Dr. Playfair and I tried to

make her stand and walk between us, she could do nothing; her legs and feet disappeared and were mere appendages to her frame. There was no power whatever of standing, she sank "in a heap" to the ground. Lying on the couch, she failed to lift the right foot from off the ground and place it on the couch. The sensibility of the limbs appeared good. The knee jerks were absolutely wanting; the vasti interni muscles were flaccid and did not respond to direct percussion; the sole reflex was preserved. There was enormous lateral curvature of the spine. This, she informed me, had not been observed until she was ten years of age. There had never been any pains. The powerlessness of the muscles of the trunk and lower extremities was very great. With all this the action of the bladder and bowels was described as normal, although there had been occasional incontinence.

The patient had a fairly good colour with a face not markedly unhealthy looking. Her manner was highly nervous and hysterical. The pupils responded well to light.

It appeared that her condition had been ascribed to hysteria, and that in an earlier stage of her illness she had been forced to try and walk up stairs. She managed this only by the aid of the banisters, never without. At that time (three years previously) she could get about a room with the help of a chair. The general health was good. I was of opinion that the case was one of Friedreich's ataxy and that it was not fitted for the Weir Mitchell treatment.

Her mother tells me that she has two sons and one daughter besides the patient. They are in very good health, able to play lawn tennis and take long walks. She herself enjoys good health, and so does the father of the patient. Neither in her family nor in her husband's is there any case of paralysis or nervous disorder so far as she knows.

As is well known, Friedreich's ataxy is usually not confined to one member of a family, so that in this instance the exceptional limitation increased the likelihood of the case being referred, as indeed it had been positively, to hysteria; but there can be no question of its nature.

A year or two ago a young man, æt. twenty-four, was brought to me on account of difficulty in walking, the cause

of which had given rise to difference of opinion. He had a staggering gait, with hesitating and slurring speech. The knee jerks were absent.

It appeared that up to fourteen months old he had been liable to "spasmodic croup," and he was two and a-half years old before he began to stand upon his feet. As a school boy he would tumble about at times and never could learn to dance. He did well, however, in his school work, and carried off prizes, the mental development being good. He went to one of the universities, but had to quit it after two years, the proctors accusing him of intoxication, owing to his gait. This was, evidently, a case of Friedreich's ataxy, but here again I could obtain no history of a family proclivity to the disease. There was an elder brother and a younger sister, both of whom I was informed were free from disease, and I could not hear of any other member of the family having been affected in a like manner.

I cannot remember (and my very imperfect notes say nothing upon the point) whether either of these patients presented the deformity of foot which has been specially described by Rutimeyer, Burg and Ormerod. My attention was first drawn to the symptom by Dr. Ormerod, who thus describes the development of the kind of club foot which is apt to occur in cases of Friedreich's ataxy. "The instep becomes prominent, and the meta-tarsals appear to be shortened; at the metatarso-phalangeal joints the toes become over-extended, and at the first inter-phalangeal joints flexed. The foot looks humpy and shortened. Below, the plantar arch (at least as seen from the inner side), is abnormally high, and the balls of the toes are very prominent. The foot also tends to assume the position of equino-varus."[1]

I remember seeing a case (which I do not now doubt was one of Friedreich's ataxy, although I failed to diagnose it at the time), in which the only complaint made by the patient was of this club foot, to which was attributed the difficulty in gait. The knee jerks were absent. It may be well to mention this, as there is always the possibility of the disease

[1] BRAIN, January, 1888.

being overlooked from the attention being directed to one prominent symptom.

There is no doubt that as a rule the fact that a patient has been observed on some occasion when she thought herself alone, to perform some movement which has been previously alleged to be impossible, lends us very important aid in coming to a diagnosis. This circumstance occurred in a case which I will briefly relate.

A lady, aged thirty-three, was seen by me at Dr. Playfair's request on June 16th, 1884. She had been for nearly eighteen years confined to her bed on account of loss of power in her lower extremities.

The patient had fallen down a flight of steps at the age of fourteen or fifteen, and shortly afterwards began to walk with difficulty and only with the help of sticks and crutches. She was seen by an eminent surgeon, who thought that she might have injured her spine and advised that she should rest. She took to her bed, and shortly afterwards completely lost the use of her lower extremities.

The patient when I visited her lay on a couch, with her legs semi-flexed at the knees and turned on their side. The feet were dropped and toes pointed. She said that she was unable to move her legs voluntarily in the least, but that they moved sometimes of themselves. She knew how they were lying without looking at them. The muscles when examined electrically were found to contract normally to induced currents. There was complete anæsthesia of both legs from just above the knee downwards—in fact of that portion of the lower extremity which would be covered by a stocking. The limbs were emaciated, the muscles being small and flabby, but there was no picked-out atrophy. The knee jerk was good on each side, and the tendo achillis jerk well marked; there was no ankle clonus. The plantar reflex could scarcely be evoked. The spinal column was free from deformity, there was superficial tenderness in the mid-dorsal region, and also in the nape of the neck. The patient gave me her left hand saying that the right was somewhat weak. The muscles of that arm contracted normally to induced currents.

The cutaneous anæsthesia was entirely confined to the legs. The patient did not suffer from any pains in the extremities; there were no bed sores. She was liable to severe dysmenorrhœa. The sight was perfect, the intelligence high, the general health very good.

I was informed that on two or three occasions the urine had been passed in the bed without the patient's knowledge. On one occasion when she had been laid up for about ten years, her maid, coming unexpectedly into her room, found her standing at the window, whence she walked to bed again. Even without this bit of history the case, it will be seen, presented no difficulty in diagnosis.

A confident opinion was given that the case was one of hysteria, and the patient was put through a course of systematic treatment. She perfectly recovered. Dr. Playfair informs me that she has remained well ever since, and leads a very busy and energetic life.

A recent experience reminds me that we must be cautious in drawing inferences from some behaviour of the patient which appears inconsistent with other symptoms. On the 8th November last I saw in consultation a lady, æt. fifty-five, of highly nervous temperament with hysterical antecedents who had been suffering for several days past from attacks of violent colic-like abdominal pain, extending at times to the lower extremities. She described the attacks as commencing with a "creeping" up the spine. In the course of time there would be a state of intense excitement with convulsive movements of the arms.

It appeared that one of her breasts had been removed in the early part of the year on account of malignant disease, and the patient was under so fixed a belief that the present illness signified a reappearance of this disease, that she could not be pacified.

The patient looked thin, but not emaciated. She lay in bed and appeared to have especially great difficulty in flexing the thighs upon the trunk; dorsal flexion of the feet was performed more easily. The muscles were very flabby. The knee jerks were absent, and there was no plantar reflex. She complained of numbness over both lower extremities

from about the level of the tenth dorsal vertebra downwards, and in this district touches were felt imperfectly. She told me that a fortnight previously numbness had commenced in the right lower extremity, and also rather to the left of the spine. At the same time there was weakness of the right leg. This had gradually, within the last few days, invaded the left lower limb, but the right was still the worst. She was sure that the loss of power and the numbness both commenced together in the right limb. Nothing wrong was to be detected about the spine nor in the abdomen.

The nurse said that the patient when by herself walked about the room quite actively, in strong contrast to the feebleness of movement shown when she was observed. In view of this, the patient's manner and history, and the absence of any sign of growth under the most careful examination, opinions had been expressed that the symptoms were probably functional, and it was with a view of helping to clear up this initial difficulty that my opinion was asked. From the fact that, whilst the wrist jerks were strongly marked (as is common in hysterical women), the knee jerks were quite absent, I had no difficulty in coming to a conclusion that the symptoms depended upon organic lesion. The opinion was expressed that she was suffering from neuritis, and this was assigned to the cauda equina. It might be due either to rheumatism, gout, syphilis, or malignant disease. The latter contingency was to be feared in the circumstances. Large doses of iodide were advised.

On Nov. 20th, there was still further loss of power, but the attacks of colic-like pain had ceased. The vasti muscles did not respond to induced currents. On Dec. 6th there was absolute paraplegia except that she could just move the toes of the right foot. The bladder was not affected. There was difficulty in swallowing and dangerous choking. Her condition became more and more grave, and on Dec. 9th she died, after an attack of choking.

There can be no doubt that she succumbed to secondary deposits of cancer.

In the case preceding that which has just been related, it will be noted that the patient had occasionally suffered

from unconscious discharge of urine. In my experience this is not at all of unfrequent occurrence in cases of hysterical paraplegia. The behaviour of the bladder has sometimes been made a crucial test in the question between functional and organic disease. It is quite certain that patients affected with hysterical paraplegia may suffer from inability to prevent discharge of urine into the bed. It is notorious, of course, that they are liable to retention. An instructive illustration of urinary troubles occurring in the course of a case which was evidently hysterical will be found in the narrative which follows.

A female patient, æt. thirty-three, was admitted into the National Hospital for the Paralysed and the Epileptic on May 26th, 1886, on account of loss of power in both legs and the left arm, of five years' duration.

In the autumn of 1879 the patient had suffered from "inflammation of the lungs," and afterwards from general weakness with "weak heart" and faintness. In 1881 her legs became very weak, and at the end of the year she could not move them. In 1883 the legs began to get stiff and since then they had gradually become worse.

In 1864 she had fallen downstairs and hurt the left side of the head. This was followed by a discharge from the left ear, and about a year later an abscess formed behind it, and there had been more or less discharge from the abscess or the ear ever since.

In 1878 she had broken "her left arm and wrist and finger," so she described it, and the hand had been useless ever since. The arm, however, and the elbow and shoulder had only been helpless since last July.

She never had severe pain in the back or limbs, and had never suffered from bedsore. There had been incontinence of urine for some months during the past year; constipation had existed since 1869. There had not been any loss of control over the sphincter ani.

The patient had suffered from scarlet fever at twelve, and diphtheria at seventeen. As regards her family history, the father as well as two of his sisters and two of his brothers and many other relations had died of consumption. Her

mother had cancer; many of her relations have had paralysis.

On admission the following note was taken by the house physician. There is complete left facial paralysis; a foul discharge comes from the left ear, copious in amount, with tenderness on pressure over the mastoid process, where there is a mark of an old scar. A short distance inside the external auditory meatus a pale polypoid mass is seen. In the left eye corneal opacities are seen in the lower half. There is no paralysis of the tongue, no difficulty in articulation or deglutition. There is aphonia: attempts to examine the larynx failed.

The left hand lies in a position of rigid flexion, the joints cannot be voluntarily moved. The patient cannot stand; there is rigidity of the legs in the extended position. Both ankles are extended and the toes over-extended on the metatarsus. There is no voluntary power over the joints. Knee jerks are equal; no ankle clonus can be produced, possibly on account of mechanical impediment; the wrist jerks are equal. There is no affection of sensation anywhere. The plantar reflexes are present, though slight. There is a little tenderness of the spine between the scapulæ; some tenderness is complained of in the iliac fossæ, especially the left. Nothing is found wrong in the cardiac and respiratory systems.

As there was no doubt, from the history and circumstances, that the case was one of hysteria, a few days after admission I forcibly extended the left fingers, which had been rigidly flexed. In doing so the skin was torn at the roots of the fingers. A splint was then applied. Next morning, when the splint was removed, the patient was able to move her fingers to a considerable extent. The legs were now also forcibly moved at the knees, and the feet actively dorsal-flexed as far as was possible. There were evident adhesions in the ankle joints, which obstructed flexion. Some of these were broken down. Two days afterwards the patient could move all the joints of her legs a little. There was still aphonia. Although the grasp of the left hand still measured 0 on the dynamometer, she was found to be using the fingers

of this hand nimbly enough for knitting. After three weeks it is noted she has now very good movements in the left hand and arm, and has considerable grasping power with it. She can move the toes very well, and bend the knees considerably, but the feet are extended at the ankle joints and cannot be flexed on the legs. There is still aphonia, but she can close her glottis for the purpose of coughing.

About six weeks later the patient was discharged, able to walk fairly well, though a little awkwardly, without help. She could use her left hand and arm perfectly well. The voice had entirely returned. There was no rigidity of any of the limbs.

This was undoubtedly a case of hysterical paraplegia, and is important as illustrating, besides the incontinence of urine, some of the trophic disorders which may result simply from long disuse of limbs. The formation of adhesions in joints, requiring forcible disruption, I have seen in other cases which were proved by the sequel to be of purely hysterical origin. There was no real muscular contracture in this case, although the dropped position of the feet suggested overpulling of the sural muscles. As soon as the adhesions were all torn through, the feet could be dorsal-flexed into a normal position. I have reason to think that this condition of joints is often regarded as strong evidence against the diagnosis of an hysterical condition, but I have convinced myself that this is an error.

Nor was there any real muscular contracture in the left forearm, although the *primâ facie* resemblance to the late rigidity so often seen in hemiplegia, was remarkable. In true hemiplegic contracture it will be observed that you cannot by any amount of force straighten the whole limb at one moment. If you straighten out the fingers, the wrist remains rigidly flexed. Bring the metatarsus into a line with the forearm by extending the wrist, the fingers will *ipsô factô* become rigidly flexed. But in this case I found, as I have often seen in other cases of apparent contracture of hysterical origin, that I could extend the fingers and wrist at the same moment, thus bringing the forearm and hand and fingers into the same plane. In doing this, however, the

skin at the roots of the fingers gave way, so that there was a transverse tear at each metatarso-phalangeal joint, shewing that there had been an adapted contracture of the skin from long disuse of the member.

The preservation of the plantar reflexes in this case was an exception to the rule, which according to my experience is of almost universal application, that in hysterical paraplegia tickling the sole produces no response. Sometimes by persevering in a very elaborate titillation the reflex is produced, and occasionally also it is easy to see that a good deal of voluntary action is expended in restraining the muscular contraction. But usually there is a simple absence of the plantar reflex, the stimulus being felt as touch only, even in persons who are naturally very ticklish. The behaviour of the reflexes in hysterical paraplegia is almost always the converse of that which obtains in tabes dorsalis—the knee jerks are exaggerated and there may be ankle clonus—the plantar reflexes absent. The exaggeration of knee jerks occurs even when there is no tendency to spasticity in the limb, which, indeed, may be abnormally lax.

In tolerably recent cases of hysterical paraplegia the limbs may be firm and round, the skin healthy looking, and the knee jerks well marked. If in these circumstances inability to extend the knee joint and dorsal flex the foot is alleged, whilst the muscles respond normally to induced currents, and there is no spasticity, the diagnosis I take it, must be safe, because one can exclude the presence of lesion in and above the lumbar enlargement. But given a partial loss of power in a limb, with a tendency to rigidity, and the difficulty of diagnosis is often enormous.

Here we enter the field occupied by cases of incipient insular or disseminated sclerosis, and these are apt to offer diagnostic problems which are sometimes of almost insoluble intricacy. There are few diseases more easily recognised than disseminated sclerosis when typically developed; there is none, I think, which may present so much difficulty when the symptoms which combine to constitute the type are almost all absent, or but very faintly expressed, obscured too, as they continually are, by very evident signs of hysteria. I

was long ago indebted to an article by A. Pitres[1] for hints regarding the insidious modes in which this disease might make its approaches, but notwithstanding this and in spite of all the care which a peculiar interest in the subject has disposed me to take, I am conscious of having made not a few mistakes in diagnosis. From my own observation, and what I have known of the practice of others, I am convinced that a very large number of cases of an early stage of disseminated sclerosis—the stage in which there are probably patches of sub-acute interstitial myelitis—continue to be diagnosed either as examples of hysteria or of voluntary simulation. The most characteristic symptom of the disease is doubtless that tremor upon voluntary exertion which Charcot has so admirably pictured in his classical account of the disease. But this, as well as the articulatory difficulty, the rigidity of muscles, the nystagmus, and the so-called apoplectiform seizures, may be absent. The symptom which is perhaps least liable to fail is an excess of knee jerk, but even this may not be present—in rare cases, indeed, there may be no response to a blow upon the ligamentum patellæ—ankle clonus may not unfrequently be unattainable. Moreover, as is well known, exaggerated knee jerks are common in hysteria, and ankle clonus also is sometimes observed. The place properly (so to speak) occupied by the characteristic symptoms of the disease may be usurped by a temporary weakness of one limb, a transitory numbness in an extremity, loss of sight of one eye, a narrowly localized atrophy of a muscle group, or some so-called rheumatic pains. It is evident that when one of these symptoms (the atrophy alone perhaps excepted) stands by itself, and is obscured by very distinct signs of hysteria, the probability of an error in diagnosis is enormous. Indeed in the nature of things it is impossible in all cases to avoid this, but the chances of error are obviously diminished in proportion as we are alive to them.

I have been able in several instances to follow up cases which presented great difficulties to myself and others, and the sequel in these has proved so instructive that I make no

[1] "Anomalies de la sclérose en plaques."—*Revue Mensuelle*, 1877.

apology for detailing them. Unfortunately they are in some instances not quite so complete as would be desirable. Nor is it at all easy to describe with the pen the peculiarity of look and manner which so strongly suggest hysteria when one is in the presence of the patient.

A patient whom I saw on only one occasion nearly thirteen years ago, exercised my curiosity very much, and I should be very glad to learn for certain the ultimate issue.

A girl, aet. 20, was sent to me by her medical attendant on June 5th, 1877. There was a bad family history. Father epileptic. Several brothers and sisters died early.

Her mother, a very intelligent woman, had prepared the following notes of her case. "Almost total loss of the use of her legs, especially the left, which is very stiff. Formerly the right leg was most affected. Sharp pains through the right knee.

"Tingling feeling in fingers, with occasional numbness in the left hand.

"Mouth sometimes slightly drawn. At such times speech slightly affected. Appetite bad. Complains of feeling swollen. Her feet are generally cold. Her habit is costive. The breath is offensive.

"Her temper is peevish, variable, and hysterical.

"The urine is thick and high coloured: it reddens litmus paper and stains linen. Its specific gravity 1013. It contains no sugar or albumen. Perspiration is profuse and offensive."

The patient was a tolerably well-grown girl with red cheeks and large pupils. She could not stand without support, and then only with great difficulty. The legs, being bared, presented a helpless appearance—especially the left. The hair on the skin was rather long. The feet were cold and clammy. In trying to reach a chair in my room she fell down on the floor. I was told that she had fallen down several times at home.

In February, 1876 (about sixteen months previously), there had been a weakness of the right leg and the left hand, of which she got the better. Again, in the autumn of the same year, whilst at the seaside, there occurred suddenly

a weakness of the right leg. But just before she came to me the left leg became the weakest. She complained of tingling in both hands (most in the left), and said that she could not write, play the piano, or draw as she formerly could. She suffered very sharp sudden pains like a knife going through the right knee. There was also a dull aching pain in the left knee—especially in walking. A well-marked feeling of waist constriction was described.

Her articulation was peculiar—the words seeming to stick in her mouth. When at my request she held out a pencil in her right hand, and tried to measure the length of a face in a photograph, the arm shook up and down, so that she failed to do it.

In the left leg the patellar tendon reflex was strongly marked. When seated, if she drew the left foot towards her at a certain point spontaneous ankle clonus of a very marked kind was set up. This also happened when she lifted her foot from the ground and held it up. Tickling the sole also gave rise to clonus.

The heart and lungs were healthy. There was no spinal curvature nor tenderness on pressure. The ophthalmoscope showed no change. There was no nystagmus. In childhood she had been rickety, it was said, and she did not walk until more than three years old.

Her mother told me that within the last year or two she had become completely changed. She had taken strongly to ritualism. Her moral character had degraded—the girl having become peevish, ungrateful, and strongly inclined to prevarication. It appeared to me that along with the hysterical symptoms there was typical evidence of disseminated sclerosis, and I expressed a distinctly unfavourable opinion as regarded the future. Arsenic was suggested, and a visit to the seaside.

A few months later I learned that she had quite recovered. But I could never get the opportunity of seeing her. In July, 1878—a year after she had been brought to me—I learned from her medical attendant, who frequently saw her socially (not professionally), that the girl seemed quite well. She could walk any distance, occupied herself, and appeared,

as far as he could judge, to have nothing whatever the matter with her.

In answer to an inquiry of mine a year and-a-half after this (two and a-half years from the date of my examination), he wrote :—"When I last saw Miss T. she was very well, but she left E—— about three months ago, and has gone to reside in D——. From time to time she has limped a little, or squinted, and then has taken the arsenic. I cannot help thinking her symptoms are due to that wonderful simulator hysteria."

Since that time I have been able to obtain no information about this patient. I have still no doubt that it is a case of disseminated sclerosis, but the narrative illustrates some of the difficulties encountered in coming to a conclusion as to the functional or organic nature of certain symptoms.

The fact that in the course of disseminated sclerosis there are apt to be periods varying in length, of almost or even complete subsidence of symptoms, though mentioned by Charcot and writers generally, is not yet, I think, sufficiently remembered and taken into account when we are occupied with the diagnosis of a case which presents difficulties. The circumstance of this girl's apparent recovery would not unnaturally lead to the conclusion that the case had been simply one of hysteria, which the consultant had mistaken, but it will have been observed that a year after the supposed recovery mention is made of occasional "limping" and "squinting," expressions which in the circumstances are of grave significance.

Before remarking on the value of some individual symptoms, I should like to record some cases which originally gave rise to a difficulty of diagnosis which has been resolved by the sequel.[1]

A lady, æt. thirty-one, was brought to me on February 7th, 1878, on account of loss of power in the lower extremities. She had been quite well, I was told, though never very robust, until two years previously, when she

[1] Many of the cases which follow could not be read at the meeting for want of time.

began to complain of numbness in the feet, and shortly afterwards in the right hand, and also of stiffness in her joints. There was also some numbness in the left side of the face and head for a short time.

At first she could not feel the ground with her feet, and then by degrees there was failure of power, so that she tottered in walking. This had gradually increased, and at the time of her visit to me she could not apparently walk across the room without taking hold of the furniture in order to balance herself.

Now and then she had a feeling of waist constriction. Sometimes there was delay in passing urine. At other times she could not hold it. The bowels were always confined. The grasp of the right hand was almost *nil*. In picking up small things from the table there were rhythmical movements of the hands. The faradaic excitability of the arms was normal. She complained of pain, or a dull weight at the back of the head; there was no noise in the ears. As she sat everything seemed going round. She had had no vomiting. Always myopic, her sight had latterly become feeble. With all this she had rather gained flesh.

With the ophthalmoscope I found the right disc more pale than the left, which, for its part, was of a dull grey hue. There was no posterior staphyloma. She could see much better with the left than with the right eye. There was no nystagmus. She had a sallow aspect. The heart and lungs were healthy. Three sisters had died of consumption.

On the right side there was ankle clonus. When she pointed the foot it was moved wildly to and fro. The patient was sent to me as a case of hysteria.

It was clearly an example of disseminated sclerosis. I did not see her again, but learned a year or more afterwards that she had become decidedly worse.

I saw, November 23rd, 1881, with Dr. Playfair, a young lady, twenty-three years of age, who could not walk without help, and the question to be determined was whether the case was one of hysterical paralysis, which it had been pronounced to be by many of the medical men who had been consulted. Dr. Playfair was inclined to think that it was

not a case of hysteria. The patient who was unwholesomely fat lay in bed. I was struck at once with the fact that she had a little horizontal nystagmus; the pupils were moderate in size; she described herself as being short-sighted. Some five years previously she had complained of her eyes, and had been advised to go to the seaside.

In 1876 she strained her left knee, and was lame in that leg for two or three months afterwards. In 1878 she had a fall on the back, and since then she had gradually lost power in both legs, in the left especially. There was no loss of sensibility. I found the knee jerks excessive in each leg and ankle clonus in the left, rather strongly marked, slighter clonus in the right. The reflex from the soles of the feet was very imperfect and suggested a hysterical condition. The legs seemed flabby and the skin was coarse and cold and inclined to the purplish tinge that one often sees in the legs of hysterical patients.

As she lay in bed with her hands upon the bedclothes in repose they were quite quiet, but when at my request she put out her right hand to pluck a grape from a bunch, the arm immediately betrayed the rhythmical movements characteristic of disseminated sclerosis in a marked form. The same symptom was noted though to a less extent in the left arm; so also, when she sat up in bed with help, the muscles of the trunk became affected with movements and the head began to shake. When put upon her feet the muscles of the trunk and extremities felt rather rigid.

I examined the eyes with the ophthalmoscope; the right disc showed no important change, but the left presented that dark grey discoloration (resembling the tint of hyperæmic grey matter of cerebrum) which I have seen now in several cases of disseminated sclerosis. My opinion was that the patient was affected with disseminated sclerosis. Dr. Playfair recently informed me that this young lady was brought back to him six years afterwards in the hope that he would undertake her case which, however, he declined. By that time the symptoms of disseminated sclerosis were very marked and far advanced.

The following case is extremely interesting. Mrs. ———

was seen by me in consultation with Dr. Playfair on March 13th, 1883. She was the mother of four children, the last child having been born a year previously. She had been ailing for two or three years, and complained of loss of power in the lower extremities. There was partial rigidity of the legs, which were not ill-nourished. The soles of the feet were very ticklish and the legs moved up vigorously when they were touched. Tendon reflex was in excess and there was ankle clonus. As she lay she was unable to move either foot from off the bed. She complained of a sense of tightness around the ribs. Examined by the ophthalmoscope the right optic disc was seen to be white and presenting that metallic glistening look which is observed in some states of atrophy.

There was a history of loss of sight of the left eye with gradual recovery, and later, of loss of sight of the right eye with gradual recovery, which had been thought to point strongly to hysteria. In spite of this, I believed from the symptoms that the case was probably one of disseminated sclerosis. She was afterwards seen by a distinguished colleague, who thought that the condition was hysterical, and she went under Weir Mitchell treatment in the hands of Dr. Playfair.

I learned only a few days ago from a friend of this lady that she "sometimes walks five or six miles to the services of her church on Sundays." Her husband's report of the same date is "Partial only, and I fear never likely to be complete recovery. Greatly improved power of locomotion, less nervousness, but a great deal remaining, and decided inability to think of much at any time."

A lady, aged 27, the mother of two children, the last being three years old, was seen by me in consultation with Dr. Playfair, on June 6th, 1883.

The patient who said that she had enjoyed good health till two years previously, did not present the aspect of illness in her face. Her complaint was of difficulties in walking and also in the sense of sight. When examined I found that she could not guide her steps rightly, and her head shook with every step that she took. She could not go through a

door straight. If she ventured to look back in walking she would fall down from a feeling of extreme giddiness. She had been numbed all over her body. The left leg appeared to her to be weaker than the right, the knee jerks were present and well marked in each leg, more in the left than in the right. There was no ankle clonus. No tremors of the limbs were noted. The wrist jerks were well marked. She did not suffer from headache, or deafness, and there was no facial paralysis. The patient's manner was highly hysterical.

It appears that she lost the sight of one eye (the left) entirely two years ago. It recovered to a certain extent in about six weeks. One year ago the sight failed in the right eye, though not to the same extent as it had done in the left. At the time of my examination she could not read or do needlework after exerting herself; she was always obliged to lie down first. After resting thus for a few minutes she was able to read a book. She read No. 2 type at four inches; the right pupil contracted to light; I was unable to be certain about the contraction of the left. Her appetite was bad: she did not suffer from any pain, but was depressed in spirits. She had lost all numbness, except in the tip of the tongue and the fingers. It appeared that two years previously she had felt numb all over the body; the numbness began in her waist, and descended to the feet, and then mounted from the waist to the throat. The ophthalmoscopic examination showed no change in the optic discs. As this patient quitted the house walking, I noticed her gait, which was staggering like that of the intoxicated.

She appeared to be very quick and intelligent, and this her husband confirmed. He said, however, that she was emotional and inclined to be sentimental. He told me that two very distinguished authorities had both considered the condition hysterical.

I wrote to Dr. Playfair that I could detect no distinct evidence of organic disease.

It seems that when the eyes failed she was taken to an ophthalmic surgeon, who examined her and found nothing perceptibly wrong. He sent her to an eminent colleague, who took a grave view of her case. This patient was sub-

mitted to systematic treatment by Dr. Playfair, who, in answer to my enquiry, writes as follows:—"I treated this case. I have a note, July 21st, 1883, to this effect: Mrs. J—— got quite well, and could walk with perfect ease, but she will not take my advice about going away with her nurse, and is going straight home, and I fear she will relapse. This fear appears to have been justified, for I have a letter from her husband, dated September 24th, 1883, in which he says: 'At first my wife was able to walk a mile or a mile-and-a-half, but she did not seem to make further progress, and latterly she has lost ground very much, and her sight is worse than before, and she can now only walk a very short distance.'" For my part I should have little doubt from the last paragraph that the case is one of disseminated sclerosis, but I have failed to obtain any more recent information regarding her.

A lady was seen by me in consultation with Dr. Playfair, November 27th, 1884. She was very deaf, fifty-three years of age. I found her lying in bed; she could not completely flex the knee joint, though Dr. Playfair had seen her do it on the preceding day. In trying to do this she lifted the whole lower extremity off the bed. The right limb was alone affected, the foot was flexed in the plantar position, apparently from semi-contraction, whether or not of the plantar fascia I am doubtful, but certainly there was contraction in the sural muscles. As she thus lay in bed, the tendo achillis was felt to be very rigid, but when I got her out of bed, seated, with her foot on the floor, this rigidity was not noticeable whilst she sat with the leg at right angles to thigh. In this position, unless by a determined effort (looking meanwhile at her feet), she brought the heel down, this part of the foot remained off the ground, but apparently she could put the flat of the foot on the ground. It was, therefore, impossible to say whether or not there was any permanent contraction of the sural muscles. With a view of helping towards the solution of this point I tested the anterior tibial muscles of the right leg, which responded normally (considering that they were smaller than the left) to induced currents.

The knee jerk was exaggerated on both sides, and there was some ankle clonus in the right leg. There was no affection of the bladder or bowel.

Her symptoms dated from two or three years back. Several years previously she had lost the use of the left arm and leg for a time, but recovered on going to Switzerland.

There was a sense of numbness in the right leg, as if it were covered (as she described it) with an extra skin of kid. The plantar reflex was good in both feet. Frequently she did not know where her leg was, and when she got out of bed it seemed quite useless and an encumbrance. It tended to wave about in a "drum-stick" fashion. She described her leg as being better than it had been, and she said that both her leg and thigh were bigger than they had been. She complained of a soreness in the left sacral region, which she felt especially on putting the right foot down.

The opinion was given that the condition was of functional character, and she was put under systematic treatment by Dr. Playfair. He reports to me, Nov., 1889: "This patient got apparently quite well. When she left me she had got rid of her crutches, sticks, &c., and could walk about. I have not since heard of her, but her nurse tells me she heard about a year ago that she was fairly well, but still could not walk perfectly." This would be four years after treatment.

The sequel as thus described, as well as subsequent experience in cases presenting the symptoms noted, make me think that I was wrong in my opinion, and that the case was one of organic disease, probably of disseminated sclerosis, which had evidently, however, derived great benefit from the treatment. I would note here that I was probably unduly influenced by the statement that several years previously she had lost the use of the *left* arm and leg for a time, but recovered on going to Switzerland. I shall have to say something more on this point later on.

A well-grown, healthy-looking lady, æt. twenty-five, of good figure and development, was seen by me in consultation with Dr. Playfair on Nov. 17th, 1886.

Until five years ago—*i.e.*, when she was twenty—her

health had been good. She was very active, particularly fond of walking, and able to walk nine miles. Then, without ascertained cause, she began to get some powerlessness of the left leg, which was at first thought to be rheumatic. It went on increasing so that ere long she could only walk half a mile. Since that time, although she had varied apparently a good deal—being sometimes scarcely able to move her legs and at others walking pretty well—she had never been able to walk two or three miles. The loss of power, which began in the left leg, shortly extended to the left arm, and afterwards the right leg and right arm became affected. It would sometimes be the right, sometimes the left lower extremity which was most weak, and so likewise with the arms. Moreover it would occasionally be the leg of one side and the arm of the other which would be co-incidently affected.

The patient walked in a tottering way. The muscles flexing the thigh appeared to be stronger on the left than on the right side. She could get up on to a chair with either leg first, but with the left better than with the right. The muscles appeared to be well nourished. Her power has varied very much from time to time. Five years ago she had a manifest squint with double vision, which lasted for several months. It was on account of the squint that she was taken to an eminent physician, who expressed an unfavourable opinion of the case. The knee jerks were exaggerated, and ankle clonus was said to have occurred, but I did not elicit any. An opinion was given that the condition as then observed was of functional character, and systematic treatment advised. Dr. Playfair treated her, and she apparently got so far well, that when treatment was discontinued there was only a slight difficulty in walking. From recent enquiry, however, it appears that now, three years later—she remains *in statu quô*, and is evidently affected with organic disease.

A lady, aged thirty-nine, the mother of one child, was sent to me by Dr. Playfair on July 9th, 1888. She complained of loss of power in walking. In Scotland, four years previously, she could walk six miles, and gradually after that

she lost power, staggered in her walk on looking up, and was obliged to hold on to something. At the same time, she said, a touch was not felt on the left side of the forehead and cheek, and the mouth was pulled on one side—she thinks the left. When examined, it is noted that her hand is steady for holding or writing; the ends of her fingers tingle. Her speech is of a hesitating clipping character, but this, it seems, she has had since the age of fourteen. Her sight began to fail in 1882; the failure in her walking powers a year or two later. The knee jerks are well marked; there is no ankle clonus. She picks up anything from the table easily and without tremor. Three years ago she could not see to read and write, but recovered the power, and for two years she could read fairly small print. Now she can hardly read minion (No. 4) at all. Both optic discs are found to be distinctly atrophic.

Dr. Playfair informs me that this patient apparently made a good recovery under systematic treatment, and went abroad with her nurse. For two and a-half months her condition remained quite satisfactory. Then one day after unusual fatigue she had a cerebral attack, the precise nature of which is left in doubt. She appears to have been quite unconscious for a considerable time, perhaps some days, and was supposed to be dying. Eventually she recovered from this and came back to England.

I saw this patient on May 29th, 1889, and examination shewed distinct symptoms of disseminated sclerosis. The cerebral attack was probably of the apoplectiform character described by Charcot as occurring in cases of disseminated sclerosis.

A lady, æt. twenty-four, was seen by me with Dr. Playfair, on May 14th, 1884, when the following notes were made. She has a good colour, large pupils, and is not unhealthy looking. She is rather, but not very thin. She rises from a chair with difficulty and stands tottering, glad to put her hand on anything to support her, and elects to have the feet wide apart, so as to enlarge the base. In walking, the gait is somewhat ataxic, with a slight tendency to over-action. There is but little power in the flexors of the thighs. The

knee jerk is much in excess on both sides, and there is ankle clonus. The plantar reflex is present and not abnormal. Complaint is made of considerable anæsthesia of the lower extremities. A touch is felt, but dimly and less distinctly on the right than on the left leg, and she cannot distinguish by the touch of her foot a Turkey from other carpet. In reply to a suggestion, she says that her legs feel as if asleep. She does not know where they are in bed without kicking against something. There is slight cutaneous anæsthsia in the fingers. She cannot pick up small things without looking. She has had cutaneous anæsthesia of the left side of the face, and indeed more or less all over her body. No bladder trouble is reported, except rather frequent micturition. There is no nystagmus.

The patient's illness dates from three years. First there was deafness, or rather sounds were heard as though at a great distance. Then followed diplopia; there was no strabismus perceptible, according to her sister's account. A surgeon said there was paralysis of a nerve, but an ophthalmic authority is reported to have said that it was of no consequence. In a week or two or more she lost the sight of the left eye almost entirely, but after some little time it returned. Now nothing abnormal is to be detected with the ophthalmoscope. She says that now the sight of the *right* eye is not so keen as that of the left.

It seems that at one time she had lost the use of both her arms. Now there is good grasp with each hand; no increased reflex is to be observed at the wrists. There are no tremors in the arms.

An opinion had been given by two eminent physicians three years previously that this patient had disease of the spinal cord, but two equally distinguished authorities who had more recently seen the case (one of whom had indeed sent the patient to Dr. Playfair) said that it was an example of hysterical paralysis.

Having been informed that three years previously the anterior muscles of the left leg had been found by a high authority to give no response to induced currents, whilst those of the right contracted well, I submitted the patient to a careful examination with electrical currents.

The application of currents to the legs gave rise to a good deal of reflex muscular action in the muscles moving the thighs. The Faradaic excitability was everywhere quite normal in the lower extremities, as also in the forearms and hands. With the galvanic current K S Z > A S Z. I gave the opinion that all the symptoms observed at that time could be explained by functional trouble, but some reserve must be felt owing to the past history.

The patient underwent a course of systematic treatment for nerve prostration, but without satisfactory result as regards her power of walking.

At her mother's request I saw her nearly four years afterwards, February 27th, 1888. She was then a good deal more helpless, but could lift the right knee a few inches, and the left, perhaps, one inch. She could not dorsal-flex either foot. With a stick and an arm she walked in a laboured manner, and the exertion to do this caused her great fatigue. Her eyes now presented nystagmus, both horizontal and vertical. There was a little waviness in the movement of the hands. Speech and hearing were good. Patient suffered from great frequency of micturition, every quarter-of-an-hour needing relief. She had no trouble with the bowel. She slept well, and her general health was good.

Tested by induced currents the anterior tibial muscles of the left leg shewed more defective response than those of the right, but on both sides the reaction was distinctly lowered. It does not need to be said that symptoms of disseminated sclerosis were now strongly pronounced.

In the case which follows a diagnosis of hysteria had been made before it came under my observation, and the girl certainly presented very much the aspect of an hysterical patient. It seems worth recording because of the sequel.

A young woman, æt. twenty-two, was admitted into the National Hospital for the Paralysed and the Epileptic on August 3rd, 1887, on account of loss of power in the hands and also in the lower extremities. There was no history of injury. She had suffered from measles when a child. About five years previous to admission she began to suffer from pains in both hip joints and weakness in the legs. Nine

months ago her knees gave way, and she complained of severe pains in the lower limbs. For three months she was able to go about with the aid of sticks, but for the last four months had been confined to bed.

For the last three years she had had attacks of loss of power in the hands, accompanied by semi-flexion of the fingers and pains.

On admission it was found that she could perform all movements of the upper limbs. No wasting or rigidity was present. She stated that she had occasional numbness and a pins-and-needles sensation in the fingers of both hands, and that she often had loss of power in the hand for a short time. No loss of sensation was observed. When asked to take hold of a pen held at a distance, there was marked tremor and inco-ordination. This was present in both upper limbs. It was with difficulty that she could drink from a cup without spilling the contents. The wrist jerks were present on both sides, not exaggerated.

She could perform all movements of the right lower limb as she lay in bed, but they were feeble. The left lower limb was flexed at the hip and knee, and patient was unable to extend fully at these joints. She was able to dorsal-flex the foot to a slight extent, also to flex and extend to a very slight degree the left hip and knee, but was unable to perform any other movements of the left lower limb. There was drooping of the toes of both feet, especially the left foot, but the patient was able to extend them. There was great rigidity in both lower limbs, especially in the left, which resisted all attempts at passive movements. The plantar reflexes were present on both sides, ankle clonus present on both sides, especially marked on the left. The knee jerks were exaggerated; there was knee clonus on the right side.

The right lower limb was subject to attacks of great rigidity in a position of extension; the left to attacks of rigidity in a flexed position. Formerly, according to her account, the left limb was apt to fall into a rigid posture in the position of extension.

There was no loss of sensation anywhere, but the patient complained that she suffered from twitchings and drawing

up of the legs, especially of the left. She also had pain in the thighs and knees. She had been unable to walk for the last three months.

When examined she was unable to stand without help, and she could not put the left foot to the ground on account of the contraction described. There was slight nystagmus and tremor of the head and neck when the patient was asked to fix her eyes on any object. Her speech was rather slow, and there was a tendency to pronounce the words in syllables; there was very slight tremor in the lips and tongue. She suffered occasionally from pain in the head, in the vertex, and also in the forehead. She described having frequently suffered from diplopia. The pupils reacted to light and to accommodation.

There was no spinal deformity and no tender spot on percussion of the spine. There was no vesical trouble, and nothing wrong in the respiratory and circulatory systems.

The optic discs were very pale. On sustained convergence there was well marked quick nystagmus occurring simultaneously in both eyes, but generally better marked in one than in the other. The pupils were not observed to vary consentaneously with the nystagmus.

The patient having been attacked with scarlet fever, was sent to the Fever Hospital, where she died. Dr. Barlow, under whose care she was admitted, was good enough to let me examine a hardened specimen of the spinal cord, which showed characteristic evidence of disseminated sclerosis.

During the first week or two of the following patient's stay in Hospital, I could not make up my mind as to the nature of her case, and I distinctly inclined to the belief that it was of functional character. She was highly hysterical; there was a history of long continued vomiting, and of loss of voice. But further observation convinced me that it was one of disseminated sclerosis—as was proved by the sequel.

A female patient, æt. forty-two, was admitted into the National Hospital for the Paralysed and the Epileptic, Queen Square, on Dec. 3rd, 1884, complaining of stiffness of both legs, being unable to walk without aid; also of pain, especially in the back, which seemed to radiate down both legs,

shooting in character. The left leg appeared worse than the right. The pains were better when the patient was up and walking about.

For the last twenty years the patient had been subject to attacks of sickness after every meal (sometimes only after one meal in the day), the longest interval of freedom from sickness at any time being 14 days. During this time the patient used to be readily tired, the legs would ache, but she did not have any special pains.

Four and a half years ago the aching and weariness of the limbs began to get worse, and the patient complained of the weight of her dress. The calves of both legs used to swell, and become shiny and hard. This disappeared in about a fortnight or so, but would occasionally recur. After this she began to suffer much from sharp shooting pains in the head and neck, relieved by lying down. The toes also at that time became very tender, and the tenderness continued for about two months, but had not returned.

Twelve months ago the patient noticed the legs begin to get stiff, but even before this her legs had occasionally drawn up when she was in bed. On two occasions during the last fifteen months, the urine has had to be drawn off.

It is noted on admission that the patient is fairly well nourished. The hands tend to turn over, and she would drop things unless very careful; the grasp is fairly good. She is not able to do fine needlework. Occasionally there is a pins-and-needles sensation in all the fingers of the right hand. She has difficulty in touching the nose with the fingers, her eyes being closed, but there are no tremors. She is able to localise touch and pain correctly in the arms; the wrist jerks are not increased. There is no nystagmus.

She has a feeling of tightness around the waist; the whole leg from the knee to the ankle feels tight as if it were going to burst. Tactile and painful sensibility is much impaired all over the legs and thighs. The plantar reflex is well marked in both legs; both legs are very stiff. Knee jerk is well marked when the legs are not too rigid, but it is impossible to get ankle clonus because of the stiffness of the legs. She is able to direct each foot pretty well to an object.

As regards the sickness from which she used to suffer, she says that she never felt sick, but food would come up at once. There was no pain in her stomach; she was obliged to get up from the table; the smell of fish would make her sick. Eight years ago for about three months she lost her voice. Her manner is highly hysterical.

This patient died in 1885 in Guy's Hospital under the care of the late Dr. Carrington. The notes of the autopsy, for which I am indebted to Dr. Gay, are as follows:—Large bed sores over the sacrum extending down to the bone, and one over the great trochanter of the left side. Abscess in each thigh, which had been opened. Brain weighed forty-one ounces. Meninges and arteries at the base presented nothing abnormal. The left temporo-sphenoidal lobe shewed yellow softening on the surface and interior extending to the corpus striatum. The whole brain was very carefully cut into fine sections, but no sclerotic patches were found. The spinal column was normal. A patch of purulent lymph was found on the posterior surface of the spinal cord in the lumbar region. The cerebro-spinal fluid was rather in excess. Patches of sclerosis began in the pons varolii, at the centre of which there was a patch on either side, and one also on the surface. They are raised, hard, semitranslucent and glistening. There is a patch on the surface of the medulla oblongata, spindle shaped, half-an-inch long and extending inwards to the white matter. There is no softening of the cord but many patches of sclerosis on the surface or more deeply.

The dorsal region of the cord was the least affected, the lumbar most. The posterior columns were most diseased, many parts between the posterior cornua and commissure being completely sclerosed. In some parts of the lumbar enlargement the whole transverse area, including the grey matter, was affected. All the other organs were healthy.

A case which was quite recently under my care in hospital furnishes so good an illustration of the difficulty in diagnosis that may be caused where a moral shock forms part of the history that I think it worth recording.

Ann B———, æt. thirty-one, widow, was admitted into

the National Hospital for the Paralysed and the Epileptic, under my care on October 26th, 1889. The following notes were taken by Dr. Taylor, resident medical officer.

Nine years ago, two or three weeks after the death of her husband (after a fortnight's illness), which had been a very great shock to her, she suddenly in one night lost power in her legs, and was unable to stand or walk for several weeks. She gradually recovered, but has never been as she was before—has never been able to run or to walk fast.

During the last six months she has gradually lost ground, not suddenly, but she is now worse than she has been during the last seven years. Her difficulty is to get her legs to separate from each other, and also in going up stairs. For the last four years there has been at times great difficulty in passing water, and sometimes she has suffered from incontinence. Patient was a strong healthy woman previous to her husband's death. On admission she complained of difficulty in walking. The condition of the arms was normal, except that a slight tremor showed itself at the end of the act in attempting to touch an object with the left hand. In the lower extremities all movements were carried out though rather feebly and with some tremulousness, especially to be noted on bending the knees.

There was a peculiar condition of the skin, of icthyotic character, which appeared to be sufficient to account for any blunting of sensibility observed. This has been present according to the patient's account, all her life. The knee jerks were greatly exaggerated. Ankle clonus was present in the right leg, and a tendency to it was evident in the left. The patient appeared well nourished. The gait was that of a person with functional paraplegia, but the legs shewed notable stiffness. When standing the legs were widely separated and became tremulous, whilst the body swayed to and fro.

Under observation in hospital this patient has continued much in the same state. There is no peculiarity of speech and no nystagmus. The ophthalmoscope shows no change in the optic discs.

I am disposed to think that this woman is affected with

organic disease of the spinal cord, and probably in the form of disseminated sclerosis, but the difficulty of pronouncing an opinion will, I think, be manifest.

I am conscious that these records do not serve to present the consultant, whose diagnosis has been exposed to the criticism of time and treatment, in a particularly favourable light. That is a misfortune for me, but not, I trust, for my audience. And I would venture, in a very modest spirit of self-defence, to say that these cases have been selected for the reason that in many instances the sequel was not that which had been expected—with the express object indeed of showing in what the difficulties of diagnosis may consist. It would have been easy enough to have brought together numerous instances in which the result proved the accuracy of the diagnosis, but that would have been little to the purpose of my address.

There can be but little doubt that of all organic diseases of the nervous system, disseminated sclerosis in its early stages is that which is most commonly mistaken for hysteria. This is evidently due especially to the following circumstances. The disease is particularly common in young females—symptoms showing themselves about the period of puberty. There is very often a history of some moral shock immediately preceding the first symptoms. It is a question well worthy of consideration whether sudden strong emotion or long continued mental worry may not be found to be important etiological factors in the disease, but this is not the occasion for discussion of the point. In addition there are few cases of disseminated sclerosis in females in which marked hysterical symptoms are not mixed up with those belonging essentially to the disease. Obviously this combination of itself causes a peculiar liability to mistakes of diagnosis. But there are other sources of error in the fact that many of the symptoms of disseminated sclerosis are supposed to suggest of themselves an hysterical origin. A sudden alleged loss of power in a limb of an apparently healthy young female, a localised numbness, or pins-and-needles sensation, complaint of loss of sight in one eye, are symptoms familiar enough as expresssions of functional

trouble. They represent equally modes in which organic disease of the kind we are discussing may make its first appearance. These local symptoms may clear off after a short time, just as would be the case if they were of hysterical origin. The girl recovers her sight, or the use of her limb, and nothing more is heard of the numbness. A little later perhaps loss of sight in the other eye is complained of; a pins-and-needles sensation is described in some other part; another limb is said to be very weak. The opinion that the symptoms are of hysterical origin may very possibly appear to be absolutely confirmed by this reappearance of trouble in other situations. Or the patient perhaps complains of weakness and stiffness in both legs, which increase so that in six or eight weeks she cannot stand. Then comes a rather rapid improvement and she recovers her power completely, soon, however, to fail again. After recoveries and relapses of this kind, the characteristics of confirmed disseminated sclerosis show themselves.

In the other sex also in persons of neurotic temperament and inheritance there may be a great liability to error. I will give an instance.

In 1882 I examined in consultation a young man with sallow complexion, large pupils, and very nervous manner. He was lying on a couch, and said that he was not able to walk much, because walking brought on a queer sensation in his legs, especially in the left leg, a pins-and-needles numbness being described. There did not appear to be any anæsthesia of the legs. His symptoms were entirely subjective. It appeared that at Easter, 1881, he played fives during a very cold wind. He was dressed in flannels, but felt it cold to his legs, and thought that he would get a bad chill. Later there was a feeling of numbness, especially in the left leg. Since then, with intervals of good health, he had had several breakdowns, which had occurred after walking too long a distance, jumping, riding, some physical strain. There had been no moral strain.

I tested the muscular power, which seemed perfect in all the groups of muscles of both lower extremities. The knee jerk on each side was much exaggerated, and there was a

slight attempt at ankle clonus, more on the right than the left side. There was, however, considerable excess of reflex in the triceps and wrist also, whilst there was no complaint of anything being wrong with the arms, so that I thought the excess in the knees might be "discounted." There were no tremors. Plantar reflex was not very strong.

The pupils contracted readily to light; the ophthalmoscope showed nothing abnormal. His tongue was covered with a thick fur, probably from the bromide and iodide which he was taking. There was a strong neurotic history on the mother's side. I diagnosed a functional disorder, and advised that the patient should go for a voyage and then resume his studies.

I saw this patient about fifteen months later. It appeared that after seeing me he went for a time to the seaside, and thence to one of the Universities, where he took his degree satisfactorily, and appeared to be quite well. Soon afterwards he walked in Scotland as well as ever. He only complained of a little numbness about the knees, equally on each side. I congratulated myself on a successful diagnosis, but this, as the result showed, was premature. Not to weary you with details, it is enough to say that this patient now presents characteristic symptoms of disseminated sclerosis.

In view of this communication I have looked through a large number of notes of cases both of hysterical paralysis and of disseminated sclerosis which have been under my care, and in many of which I have been able to learn the issue. As regards the value of individual symptoms in enabling us to differentiate organic disease from functional disorder, I find that whilst there is still much to be desired, there are points in which experience appears to speak with a fair amount of distinctness.

As a rule, though this is not without some notable exceptions, the class of hysterical paraplegia is not difficult of diagnosis by those well acquainted with the symptoms and course of organic disease, the surrounding circumstances, and especially the contradictions palpable in the symptoms leaving one usually in but little doubt. I need not dwell upon these before my present audience, but would remark

that the attitude and conditions of the lower limbs may vary exceedingly. The limbs are most often in a state of perfect flaccidity, a condition of spasticity being comparatively rare. The feet are frequently "dropped." After long disuse it will not unfrequently happen that there are strong adhesions in the joints. I have already referred to this, and to the pseudo-contracture due partly to this fibrous ankylosis and partly to contracture of the skin—not of the muscles. Hysterical paralyses are most often complete. The loss of power in disseminated sclerosis is very rarely (except in advanced stages) more than moderate. I cannot help thinking that the view still generally held that a shifting of loss of power from one limb to another (such as that which I have described) is really characteristic of hysteria is quite an error. The hysterical woman who has lost all power in her legs, will, it is true, very often later on (whilst still paraplegic) lose the power of one arm, usually the left; but I have not found that she is prone to lose the power in a limb, then recover it, and then lose it in another. It seems to me that the idea of this shifting of powerlessness being strongly suggestive of hysteria has arisen from the mistakes in diagnosing as hysteria cases of disseminated sclerosis, which must have been continually occurring before the latter disease had been differentiated. No doubt the hysterical are prone to changes of disorder; at one time, for example, losing the use of a limb or limbs, with or without profound anæthesia, at another losing the voice, or closing one eyelid, or contracting a limb, but the shifting about of a state of more or less powerlessness, which we see in disseminated sclerosis, appears to me to be *sui generis*, and should, I am disposed to think, save us from error. And equally so with the occurrence of numbness or pins-and-needles sensation, sometimes at one part and sometimes at another, which, if my notes do not betray me, points with considerable distinctness to disseminated sclerosis.

There would appear to be a little more difficulty in regard to the impairment of sight in one eye to which I have referred. The ophthalmoscope perhaps shows no change. But we shall find, I think, that the hysterical patient as a

rule, when loss of sight of one eye is in question, is quite blind on that side, whilst the patient with sclerosis has only more or less obscurity of vision. I cannot call to mind, since I have been better acquainted with disseminated sclerosis, any case of simple hysteria in which first one eye lost some amount of vision for a time and recovered, and afterwards the other eye behaved in a similar fashion. So that this symptom I should now take to point with considerable force to disseminated sclerosis. When the ophthalmoscope shows atrophy of disc (and it is remarkable in what a large proportion of cases of disseminated sclerosis some atrophy is to be found—in some a stage of hyperæmia preceding it) my experience would teach me that a diagnosis of functional disorder must be discarded.

The same must be said of nystagmus, a symptom of peculiar value when combined with others about which there might otherwise be some doubt. It is necessary, of course, to remember the possibility of chronic alcoholism producing a temporary nystagmus, but this chance of error ought not to be difficult to avoid.

We next come to the tremor on intentional movement, upon which I am disposed to place a diagnostic value higher than that possessed by any other symptom of disseminated sclerosis. Looking back many years I can remember observing numerous cases which presented this symptom at a time when I used to feel very great difficulty in the differential diagnosis which we are considering. I cannot call to mind one which the sequel proved was simply functional. It is true that in the hysterical we not unfrequently see a clumsiness of movement of the hand when directed towards an object, which is somewhat liable to deceive, but observed carefully it will be found that this is rather of the nature of ataxy than a rhythmical tremor such as is found in sclerosis.

There is also another variety which is worth noting. The patient asked to touch an object with her finger, does so without difficulty or hesitation, but when the finger has rested upon the object for two or three seconds the arm becomes affected with somewhat rude tremors. This is in striking contrast with the tremor which affects the arm in disseminated

sclerosis, as the patient brings the finger near the object, tending to cease when it is attained.

There may be very fine and rapid tremor only when the patient stands upon the feet, ceasing when the sitting posture has been assumed.

On the coarser semi-convulsive movements, twitchings, jerkings, and grimacings not rarely met with in hysteria I do not dwell. They could not for a moment deceive any one acquainted with the subject.

Localised atrophy of muscles with loss of electrical reaction is well known to occur sometimes in the course of disseminated sclerosis, and in a case otherwise open to doubt its presence is undoubtedly of the highest value in determining the organic nature of the disease. But I do not think it is generally known that the localised atrophy may behave like the temporary powerlessness of a limb or limbs, or the shifting numbness. I have seen several cases of disseminated sclerosis in which atrophy of some muscles, with loss of electrical reaction, has cleared off entirely, to be succeeded some time afterwards by a similar lesion in another or the same part.

It is scarcely probable that disseminated sclerosis is a new disease. Little more than half a century has elapsed since it was first figured by Cruveilhier in his "Atlas d'Anatomie Pathologique," and twenty-two years ago Charcot expressed his belief—a well founded belief, I have reason to think—that the disease was not known, that is to say not recognised, in England. It is practically indeed to Charcot that we owe our acquaintance with the disease, from the admirable summary of its clinical and pathological features published by him in his earlier lectures. We all know what a length of time it takes for a disease, however excellently pictured, to fix its features so firmly in the minds of medical men generally as to make the diagnosis of it come readily to those who have not gone out of their way to seek examples of it. And this, which is true of most forms of organic disease, is from the nature of things most marked in reference to disseminated sclerosis. In that disease the infinite irregularity in the situation of the essential pathological

lesions creates difficulties of recognition beyond those to be met with in any other example. This being the case it is not surprising that the symptoms, characterised as they are by frequent remissions, should, in the absence of other explanation, be set down to the vagaries of hysteria. The almost constant admixture of circumstances pointing to an emotional origin or accompaniment of these symptoms greatly increases the likelihood of this confusion occurring. As I have before remarked, it appears to me reasonable to conclude that many symptoms which have come to be considered characteristic of hysteria will, if examined by the light of improved knowledge and experience, be relegated to disseminated sclerosis. The same principle holds good, as I have already shown, in reference to atrophy of the iliaco-psoas muscles, and to Friedreich's paralysis, not to mention others. But in none so much for the reasons mentioned as in disseminated sclerosis. There is a point of great interest to be worked out in regard to the possibility of hysteria—a disease of which the pathology is unknown—merging gradually into disseminated sclerosis. We are quite in the dark on this point, and this is not a fitting occasion on which to do more than advert to it. What is the connection between disseminated sclerosis and hysteria? It seems impossible to doubt, in view of the marked and almost constant occurrence of hysterical symptoms in the earlier stages of the disease, and the preponderance of cases affecting the female sex, that there is a connection of some kind, but in what that association consists I for one cannot pretend to say. No more interesting and important subject than this could engage the attention and study of our Society. The result, if one may venture to hazard an opinion founded on the experience of that which happens in regard to other diseases, would almost certainly be to refer to an organic origin many symptoms which we now term hysterical. The figure of Hysteria shrinks in proportion as the various forms of organic disease acquire greater solidity and sharper definition.

OF THE VISUAL AREA OF THE CEREBRAL CORTEX, AND ITS RELATION TO EYE MOVEMENTS.*

BY PROFESSOR HERMANN MUNK (BERLIN).

Translated from the original by F. W. Mott, M.D.

THE more vigorous the opposition which my statements regarding the visual areas of mammals have met with, and the longer the interval that has elapsed before it has been overcome, so much the more certainly, as I believe, have my views obtained a stable foundation. One important point only may be looked upon as an exception to this statement. Although it is generally agreed that to each visual area in the monkey (and in man) the corresponding halves of both retinæ correspond, and that in the dog the correspondence of each visual area is with about the lateral fourth of the retina of the same side, and the mesial three-fourths of the retina of the opposite side, the intimate connection which I have further made out between the retinal areas on the one hand and the cortical areas of the occipital lobe on the other hand, together with the so-called projection of the retinæ on the visual areas, still remains for the most part unconceded. The object of the present communication will be to further the acceptance of these views. I obtained the first insight into the subject in an early stage of my experiments. Having extirpated a suitable and not too small portion of the cortex of one occipital lobe, I found that visual disturbances occurred which were only to be explained by supposing that a second blind spot had been produced in the retina.[1]

* This paper, describing experiments carried out in conjunction with Dr. Ogregia (of Bucharest), was presented to the Royal Academy of Sciences, Berlin, on the 16th Jan., 1890, and is published in the Proceedings of the Academy.

Accordingly loss of perception corresponding to a certain part of the light-receiving elements of the retina always follows extirpation of a certain part of the cortex, and it cannot, as I have remarked, be explained otherwise, than by supposing that the central elements of the visual areas, in which the optic fibres end, and the perception of sight takes place, are arranged in a regular and successive manner like the sensory elements of the retina from which the optic fibres originate, in such a way that neighbouring retinal elements always correspond to neighbouring perceptive cortical elements.

These extremely delicate observations were, however, rendered very difficult because the disturbances were of a transient nature and the animal soon learnt to neglect the new blind spot. I have, therefore, in a later systematic research on dogs, removed large pieces suitably chosen from a visual area, alternating the anterior and the posterior, the mesial and the lateral halves and so on—and the disturbances of vision, now lasting long enough for observation, have enabled me to determine more accurately the connections of the retinæ with the visual areas.[2] According to the results of this research the retina may be assumed to be projected upon the visual areas in such a manner that the upper retinal zone corresponds to the anterior border of the visual area, the lower retinal zone to the posterior border of the visual area and the mesial retinal zone to the mesial border of the visual area. Finally, in so far as the retina of one side and the visual area of the other belong in the main to one another (in the dog) so does the lateral border of the retina correspond to the lateral border of the visual area (*vide* diagrams).

In conclusion, I have combined with similar partial extirpations of one visual area in dogs, total extirpations of the other, so that nothing but a remnant of one visual area remained intact).[3] These researches were most difficult to perform, but, when successful, the above determined projection was recognizable in the clearest and simplest manner. I have not made investigations to the same extent in monkeys. In transverse sections through the occipital

lobes the cortical convolutions (contrary to their great simplicity in the dog), here show a considerable degree of intricacy; and this has given rise to difficulties which could not be overcome. After numerous different partial extirpations, I have, therefore, contented myself with establishing the fact that, as might be supposed, *in principle*, the same projection exists in monkeys as in dogs.[4]

During ten years no repetition of my investigation has been forthcoming, and projection has been regarded by some as a great discovery, and by others almost as a fable. It must, indeed, seem of little worth to those who altogether dispute the dependence of the simplest visual perceptions upon the visual areas of the cortex; and it is perfectly natural that they should have failed to recognise such slight support, which even the larger extirpations in the area of the occipital lobes, have offered for the projection theory.

And as for pathological cases in man, whilst they have been forthcoming in numbers in support of my other statements in reference to the visual areas, they have seldom been favourable to the theory of projection, less by reason of the rarity of the cases than of the difficulty of observation.

Only one experimental result at last appeared in favour of projection. Of two monkeys from which Messrs. Sanger Brown and E. A. Schaefer had removed both occipital lobes, one was rendered permanently but the other only partially blind; in the latter a small part of the field of vision was left so that objects held above the level of the eyes were seen; and it was shown that in the latter case the extirpation on the under surface of the hemispheres had not extended as far forward as in the former case.[5] Meanwhile results confirmatory of projection have frequently occurred, unsought by me, in my numerous extirpations of the visual areas, in cases where the intended total extirpation of both visual areas had not succeeded on the one or on the other side. But I had not hitherto been able to discover any simpler or more easily demonstrated mode of experimentation than those that I have described.

Another road has recently again been opened up in the

direction of projection. Mr. Schaefer[6] has found that associated movements of the eyes are produced in monkeys by stimulation with the induction current of *all points of* cortex of the occipital lobes. Both eyes always move towards the side opposed to the lobes stimulated; and at the same time downwards if the excitation takes place in the upper (or anterior) and upwards if it takes place in the under (or posterior) zone of the occipital lobes: in both cases the movement (upward or downward) is strongest when the electrodes are placed on the mesial surface of the lobe or in its neighbourhood.

The intermediate zone, from which pure lateral movements were obtained, is externally wide, internally narrow, so that there is only a narrow portion on the mesial surface of the lobe.

The upward movement of the eyes was generally accompanied by an elevation of the eyelids, the downward movement by a depression, but these movements of the eyelids were as irregular in the experiments, as the changes of the pupils which sometimes accompanied the movements of the eyes. As far as could be observed the visual axes always remained parallel, when one hemisphere only was stimulated. Once, when the electrodes were placed on corresponding points of the mesial surfaces of both hemispheres, a feeble convergence of the visual axes occurred, but this result was not marked or constant enough to permit of much importance being attached to it.

We assume, therefore, says Mr. Schaefer, that these various movements of the eyes are the result of the accompanying appearances of subjective visual sensations, which the stimulation causes; and that the movements take the directions towards which those sensations are projected. Thus these experiments point to a certain connection between the parts of the cerebral visual area and of the retinæ, a connection which may be stated as follows:— First, the whole visual area of one hemisphere is connected with the corresponding lateral half of both retinæ, the upper zone of the visual area of one hemisphere is connected with the upper part, the lower zone with the lower part, the in-

termediate zone with the middle part of the corresponding lateral half of both retinæ. So far this is a perfect confirmation of my investigations. However, Mr. Schaefer goes further than this, and continues in the following manner :—"We may represent these relations diagrammatically by supposing the visual areas of both hemispheres to be united within the great longitudinal fissure and imagining each retina to be expanded and projected in its natural position upon the united area. If this is done, it is at once seen that the identical points of the two retinæ correspond with one and the same spot of the cerebral cortex; that the upper half of each retina falls upon the upper half of the united area, and the lower half of each retina on the lower part of that area; that the inner half of the one retina and the outer half of the other, each fall upon the same hemisphere, and that a vertical zone including the centres of both retinæ corresponds with the mesial part of the united area. This would connect the parts of the retinæ which are concerned in direct or central vision with a part of the mesial surface of the occipital lobes, probably including a portion of the quadrate lobules. This scheme of connection between the retinæ and occipital lobes differs in several points from the well-known scheme formulated by H. Munk (Ueber die Functionen u.s.w.: 5te Mittheilung) as the result of extirpation experiments, chiefly upon dogs, but partly upon monkeys. The most important differences relate to the extent of the visual area, and to the part of the occipital cortex which is connected with the central part of the retina (macula lutea). The visual area (Sehsphære) of Munk is *confined to the occipital lobes* and the maculæ luteæ *are connected with the middle of the convexity of those lobes.* Consequently if the middle of the convexity of both lobes is removed central vision should be abolished. But contrary to Munk, I have not found this to be the case in monkeys. Indeed, it was the smallness of the effect produced by this bilateral lesion which first led me to doubt the correctness of Munk's scheme. On the other hand, in one experiment which I have performed since obtaining the above results from stimulation of the cortex, there was a marked defect of

central vision, produced by a bilateral injury of both mesial surfaces. Complete and permanent abolition of central vision did not result, but it was found, post-mortem, that the lesion did not involve as much of the mesial surface of the visual area, especially upon one side, as was intended at the operation. It will be necessary to follow out the question more closely by the extirpation method, and this I intend in the near future to attempt; but the difficulty of arriving at reliable conclusions from the localized extirpations of the visual area, is enormous; for the animals soon acquire the habit of compensating for local defects in the visual field by rapid movements of the eyes so as to baffle all attempts to determine the existence of such defects. I believe, indeed, that to arrive at detailed conclusions, we must await the results of perimetric observations in cases of cerebral lesion in the human subject, and that these experiments upon the visual area of the monkey are chiefly of value as affording indications of the kind of results to be looked for in man."

From this it would appear that Mr. Schaefer had mainly to do with results which were at variance with my conclusions concerning projection. The points of variance are, however, of very slender importance. I have nowhere stated that the maculæ luteæ of the ape are connected with the exact middle of the convexity of the occipital lobes. In my communication of 1880, quoted by Mr. Schaefer, I only associated the maculæ luteæ with *that* part of the cortex, which occupies *about* the middle of the convexity of each occipital lobe.[8] But in a communication of the year 1881[9] which has escaped Mr. Schaefer's notice, I have laid stress upon two experiments upon monkeys as "very interesting new experiments" concerning the cortex corresponding to the maculæ luteæ—two experiments which at least counterbalance those of Schaefer—in one of which, the convexity of each occipital lobe was extirpated about the middle, in the other a piece of the same size, situated further back and somewhat more internally, was removed.

In both, the monkey fixed its eyes *after* the operation in the same way as before, but in the latter case the visual disturbance was considerably greater than in the former;

hence I have concluded that the cortex corresponding to the "fovea centralis" is situated in the posterior half of the convexity of the occipital lobe on each side. The entire difference between us amounts practically to this: I have laid more stress on the removal of the posterior than of the inner surface, and Mr. Schaefer accentuates the removal of the inner surface. We both recognize that little importance is to be attached to the difference, since the limited number of our experiments has not permitted an accurate localisation of the cortex in question to be made.

If Mr. Schaefer, however, merely bases his scheme upon the inward displacement of the visual area, so that he unites the visual surfaces of both hemispheres on the great longitudinal fissure to a single one, and projects each retina in its natural position on this visual area as a whole —then his conclusion is not only unsupported by facts, but is demonstrably erroneous. For he quite overlooks the convolution of the cortex of the occipital lobe of the monkey, especially the *double-layer* of the cortex parallel to the surface, with the portion on the mesial border of the lobe covered in, so that the mesial edge of the cortex does not correspond to the mesial border of the lobe; he forgets that important and dissimilarly placed areas of both retinæ do not take part in binocular vision at all: he takes no account of the differences which man, the ape, the dog, &c., present in binocular vision; finally he disregards the results[10] which unilateral extirpations of halves of the visual areas of monkeys have afforded.

Taking into consideration all these points, which Mr. Schaefer has neglected, we can only maintain that *in principle* the same projection exists in monkeys and man, as in dogs, both in the vertical position (which as we have seen, Mr. Schaefer himself upholds) and in the horizontal position, and that the identical points of both retinæ do not correspond to the same points of the cerebral cortex; but the external half of each retina is associated with the outer half of the visual area of the same side, and the inner half of each retina with the inner half of the corresponding visual area of the opposite side. Moreover, I have observed

in an ape, in which the intended removal of the occipital lobes was not completely successful, a condition that I have often found in dogs. The ape was totally blind in the left eye, whilst sensibility to light still remained in a very limited part of the mesial and lower portion of the retina of the right eye.

Although I have not been able to pass over in silence Mr. Schaefer's difference of opinion, still at the present moment when the theory of projection has not been generally accepted, the differences between us are not so important as our points of agreement. Nevertheless Mr. Schaefer's inference, as it stands, cannot be considered as a new proof of my discovery of projection, because, to him, the supposition is based upon electrical irritation of the cortex producing sensory impressions, a theory which was indeed assumed by Dr. Ferrier, but up to the present time has remained without proof, and even without probability.

To this is to be added that no great weight can be attached to Mr. Schaefer's inference, because it only contains a further development of a point which is contained in his second assumption, namely, that the movements of the eye take directions towards which the visual sensations are projected. Nevertheless, if one is not to assume that the coincidence is altogether accidental, it must undoubtedly be accepted as affording a support for the projection theory, and as proving a close connection between the eye movements and projection.

I have, therefore, in order to clear the matter up, undertaken a series of experiments in conjunction with Dr. A. L. Obreggia, of Bucharest; choosing dogs for the purpose, because in these animals the cortex of the occipital lobes is only slightly convoluted, and therefore more easily got at, and the visual area is known better than in other animals. The dogs were placed under ether during the time of exposure of the brain, but the observations were made without any narcosis. Herr Obreggia will present an account of these experiments in full detail elsewhere, and I shall here restrict myself to a review of the experiments and discuss the results. For the topography of the brain I would refer

to the description of the dog's brain which I gave in my fourth communication,[11] January, 1878, and which has since found a wide circulation (fig. 3).

I count the convolutions of the convexity from the great longitudinal fissure, so that the fourth convolution surrounds the fissure of Sylvius.

As Mr. Schaefer's very serviceable experiments on monkeys lead us to anticipate, stimulation of the visual areas of dogs with an induction current brings on associated movements of the eyes towards the opposite side, both the eyes at the same time moving downwards if the anterior zone is stimulated, upwards if the posterior zone of the visual area is stimulated. The intermediary zone from which purely lateral movements originate, is but narrow; generally not half so wide as the spot A (fig. 4) of which about the middle part falls in this zone.

The upward movement is produced most markedly by stimulation of the second convolution, and it diminishes by approximation of the electrodes to the great longitudinal fissure: the downward movement shows, with the same change of position of the electrodes, no decrease, but often an increase.

The strength of the current which was found necessary for successful stimulation, is about the same as that required for the other well known cortical centres for the movements of the extremities; but for the upward movement it is somewhat less than for the downward movement. With the ordinary eye-movements are often combined movements of the upper eyelids and dilatation of the pupils, the further consideration of which need not here detain us. If one stimulates the cortex somewhat beyond the anterior border of the visual area in the region F, or beyond the lateral border of the visual area in the auditory centre B, then the eye-movements cease, if the strength of the current be not increased. These movements, which as we have seen are undeniably the results of locally restricted stimulations of portions of the visual area, are by no means slight, being sometimes even very considerable, especially on the posterior zone of the visual area. That the movements

have nevertheless escaped observation, and that even experimentors who have directed their special attention to the subject have stated that what are really the most anterior portions of the visual areas are the posterior limits for the production of the eye-movements, is not surprising to any one who considers the evolution of the study of stimulation of the cerebral cortex; it would be vain to consider all the possible causes of discrepancy. It may, indeed, be looked upon as a lucky circumstance that the correct knowledge of the subject was delayed. For had not the cerebral cortex been divided into an anterior motor and a posterior non-motor part, we should not have had the Fritsch-Hitzig experiments, and the later-acquired insight of the perceptive centres of the cerebral cortex would have met with still greater opposition than was actually the case, by reason of the short-sightedness which made all spots of the cortex that gave rise to movement when stimulated, into motor centres or connected with a great motor or psycho-motor area.

The question now arises how can we adapt these later experiments to our hitherto established knowledge of the visual centres? We may see our way to this, if we recollect the results which the total extirpation of both visual areas entails. The animal is perfectly blind, but his eye-movements are intact, the so-called voluntary, as well as the involuntary; excepting, of course, the movements which are entirely dependent upon vision, which *must* be absent in a blind animal. In the course of years I have often repeated this experiment, and I have recently convinced myself that in cortical-blind dogs and apes the eye-movements persist, even when the animal remains at perfect rest, and when every external influence is removed from his remaining perceptive faculties. The visual area has nothing whatever to do with those eye-movements of the animal which are independent of sight, neither do these movements result from excitation of the visual areas nor does the path of conduction from the place of their excitation to the periphery lead through the visual centres, consequently the eye-movements which the electrical irritation of the visual area—

induces, only correspond to particular eye-movements of the animal which are the results of visual perception.

The following offers itself as the most obvious inference. Outside the visual area and in the so-called motor area —my tactile sphere—are two spots on the cortex, the electrical stimulation of which causes eye-movements, one a spot situated in the anterior part of region F, the other, in the anterior part of region H, and accordingly as one or the other spot is excited, so will the eye muscles of the animal which are set in action, give rise to particular movements, just as by stimulation of neighbouring spots C. and D. on the cortex, arm or leg muscles will be brought into action. Therefore, if an animal makes a movement in consequence of having seen anything, it must be concluded that the excitation conducted through the optic nerve fibres to the visual areas, is transferred by associated fibres which connect these same areas with the tactile areas, at one time by this set of associated fibres, at another time by that—according to the kind of movement produced, and so through certain association fibres the excitation reaches the spots C. or D. if arm or leg movements occur: through other associated fibres the spots F. or H. if eye-movements take place. In analogous fashion the eye-movements are obtained in our case by excitation of the visual area with induction currents, since the excitation produced by the electrical current spreads to the centres F. and H. by associated fibres which run from the visual areas to those centres; whether excitation is originated by the current in the central elements of the visual area, or in the fibres themselves where they terminate in the visual area.

But against this conception the objections will naturally occur, that eye-movements alone (with at the most associated movements of the eyelids and the iris) follow the electrical stimulation, never movements of any other category, *e.g.*, arm or leg movements. No doubt an explanation of this might be found in the following consideration. It is held on good grounds, that although the excitation of a portion of the central nervous system comes into relation with different paths, nevertheless, the resistance which is

offered to its spread along the different tracts is different; and, other things being equal, the more worn certain paths of conduction are, *i.e.*, the more frequently they have already been traversed by the excitation, the less the resistance they would offer. Among all the tracts concerned in associated actions between the visual perceptive centres and the tactile (psycho-motor) centres the least resistance would probably be found along the paths for the eye-movements, since these movements are associated with vision, more often than any other. For this reason the above proposition concerning the eye-movements observed in our researches might be accepted because in these experiments the currents which were employed were very weak, in fact, scarcely more than necessary to induce movement, and the excitation was confined to as limited an area as possible.

But, if this explanation were correct, stronger stimulation should lead to movements in the other parts, the paths to which offer greater resistance, and this is never the case. After the eye-movements have come under observation, even when the electrodes are placed on the anterior portions of the visual areas and still more obviously when they are placed on the middle or posterior portions of these areas, the induction current can be very considerably strengthened before epilepsy is induced by the too powerful and ever extending excitation, and up to that point eye-movements alone occur. It must therefore be acknowledged that unless an entirely arbitrary position is taken up the eye-movements occupy an entirely special situation which could not agree with our hypothesis. A further test disproves the hypothesis by a different method.[12] If a frontal section be made in the anterior zone of the visual area through the hemisphere, such as I have lately recommended for the extirpation of the visual area,[13] only deeper, so that it opens the ventricle, and if its lower end is carried horizontally from the edge of the corpus callosum to that point in which, according to my representations, the anterior and the lateral margins of the visual area join—then, as long as the electrodes are not placed in the neighbourhood of the cut surface—electrical stimulation of the visual area produces the same movements

of the eyes after the section as before. And it makes no difference if a second section be made dividing white substance and cortex extending in a vertical continuation from the lateral or outer end of the first one, outwards in front of the descending horn of the lateral ventricle, along the convexity of the hemisphere, as far as the tip of the temporal lobe, but sparing a layer 1-2 mm. thick, lying outside the optic thalamus. This layer contains the sagittal fibres of the occipital lobe.[14] According to all that we know about the position of the associated fibres, the paths for associated actions have been interrupted partially by the first section, and completely by the combined sections. These tracts connect the visual centres with the cortical centres in F. or H. hence these cannot take part in movements of the eyes when these movements arise by electrical stimulation of the visual area.

No significance can be attached to the commissural fibres which pass out of the visual areas into the corpus callosum, because no idea of the process could be thus conceived, which would not at once prove untenable. Besides it is only necessary to cut through the posterior part of the corpus callosum lengthwise, either in the uninjured brain or after the before-mentioned sections have been performed, to observe that excitation will still produce movements of the eyes. Consequently, the conclusion remains that these eye-movements come to pass because the stimulation brought about by the electrical current extends to the radiating fibres of the corona radiata which go to the sub-cortical parts of the brain, and that the excitation originates in the central elements of the visual area, or in the radiating fibres where they proceed from the visual areas. The following experiment shows that this is the case.[15]

If a horizontal section such as I have recommended for the extirpation of the visual area be made through the hemispheres at the lateral margin of the visual area, the eye-movements will then no longer appear on stimulation of the visual area. The experiment can be made at any time, but it is best performed on the still uninjured hemisphere. The loss of blood and disturbance of the circulation of the

occipital lobes cannot be made responsible for the result since the loss is, as a rule, much less than with the frontal section before described, or when several successive sections have been made. A very interesting variation of the experiment may be performed, as follows: If, on the uninjured brain one directs the scalpel, not obliquely *upwards*, as was desirable for the extirpation of the visual area,[16] but obliquely *downwards*, so that its point tends towards the under surface of the corpus callosum: then the associated fibres, such as the callosal, are preserved, while the radiating fibres are cut through, and upon excitation the eye-movements are found to have disappeared. A hæmorrhage into the ventricle is not of much importance in this experiment, for a similar hæmorrhage with the frontal section, or with its extension lengthwise, does not affect the result.

To our previous knowledge of the visual areas, as central organs for vision, is therefore now added an insight into certain connections which the visual area forms with other central organs, viz., those connections which adjust the movements resulting from vision. It may be assumed, in connection with the accomplishment of these movements, that the excitation is transferred from the central elements of the visual area through associated fibres, to other cortical areas and thence to lower (sub-cortical) portions of the brain. We now know that the corona radiata of the visual area, contains, besides the optic nerve fibres, the excitation of which transferred centrally towards the visual area produces vision, also radiating fibres, the excitation of which, transferred towards the periphery from the visual area to lower (sub-cortical) parts of the brain, occasions movements, but that these are simply eye-movements the result of vision (with accompanying eyelid and such like movements), and that all other movements which are the result of vision, require the interposition of associated fibres and other cortical areas. Our experiments do not exclude the possibility that eye-movements are brought about by the latter mode as a result of visual excitation, but it is plain that certain eye-movements are preferred before all other movements which are the result of vision, in that

they are brought about in the nearest and shortest way through radiating fibres of the visual area.

There can be no doubt of the kind of eye-movement so preferred. My last communication gave me occasion to call attention to the way in which retinal and visual perceptive reflexes are to be distinguished in animals. The contraction of the pupil when light enters the eye is a retinal or optic reflex—a common reflex action for which light perception is not required, and which is brought about by means of lower portions of the brain, without the cerebral hemispheres. On the contrary there are, as I have remarked, other visual reflexes, sensory reflexes, which are effected by the action of the visual centres, *e.g.*, when—without the co-operation of the attention or judgment—the eye blinks on approach of the hand, or the animal avoids an obstacle by moving. For these visual perceptive reflexes the excitation must, we are now able to add, take a course from the visual areas through the associated fibres to other cortical areas, and through their radiating fibres to the lower centres. But the examples given, as well as the retreat from the slash of a whip, the cowering at a stone being thrown, the parrying of a blow with the out-stretched arm, and the like, are visual reflexes of a higher order; reflexes which with all their manifold variety, have this in common—that they are not innate, but are evolved; and that for the commencing of the movements—involving co-operation of attention and deliberation—visual and other representations must arise. Besides these there is, we find, yet a third kind of reflex in animals which stand as it were midway between the two first kinds, viz., visual reflexes of the lowest order, which are innate, and presumably never have psychical visual representations, but only have to do with the perception of light or other simple visual effects: such are the involuntary movements, which cause the eyes to wander, before being fixed upon an object which was previously indistinctly seen. It must be these reflexes (exclusively eye-movements that are the immediate and direct result of vision) for the performance of which the excitation, conducted by radiating fibres to the visual areas, proceeds again directly by radiating fibres to the lower (sub-cortical) centres.

It may be observed that we have thus obtained an insight into the course of the nerve tracts which pass out from the visual areas, without having fixed our attention upon the consequences of electrical excitation of the visual areas other than the mere appearance of eye-movements, and without having found it necessary to decide whether the excitation has arisen in consequence of electrical stimulation in the central elements of the visual areas or in the associated fibres or in the radiating fibres which pass out of the visual areas. But it stands to reason that the eye-movements resulting from stimulation of the visual areas, correspond as before stated, not simply to those eye-movements of the animal which are the result of vision, but more especially to those particular eye-movements which produce "wandering vision" and fixation of the eyes upon an object previously indistinctly seen. From the direction of the eye-movements observed on excitation of the visual area, it is therefore to be concluded that to each visual area belong the portions of both retinæ of the corresponding side which are on the same side of the maculæ lutæ, and that to the anterior, middle, and posterior zones of the visual areas respectively belong the upper, middle, and lower quadrants of those portions of the retina. Thus the theory of projection to which by the help of his assumption Mr. Schaefer has arrived in the monkey, fits in with the results obtained by our method upon the dog.

Nevertheless that result is only free from objection, if one recognises that the visual nerve fibres after their entry into the visual area, are connected directly and immediately with the central elements which serve for the perception of light; and indeed this is the simplest and most natural of all considerations which I have hitherto brought forward relating to the visual areas. This point, however, might be disputed, for it might be assumed that the visual nerve fibres enter upon their nearest connection in the visual area with the central elements of the ordinary kind, such as the lower portions of the brain and spinal cord contain; and that on the one hand connecting fibres extend out of these common reflex centres into the central elements serv-

ing for the perception of light, while on the other hand the radiating fibres of the visual areas arise out of them and conduct the stimulation towards the periphery. The results of electrical stimulation would then by no means correspond to the eye-movements of the animal resulting from vision, and accordingly would not serve as a proof of projection, but they would merely indicate (no matter whether they rested upon excitation of the common reflex centres or of the radiating fibres) that both the visual centres and their respective zones stand, for purposes as yet unknown, in different relations to the different eye-movements. To meet this objection we must again turn our attention to stimulations of the visual centres.

Roughly regarded, the eye-movements which I have above described, are the usual and regular results of stimulation; and if here and there an anomalous result occurs, yet in a general survey of the visual areas, one is satisfied to ascribe it to a spontaneous movement of the animal or to some other uncontrollable circumstance which one must always assume to occur in this kind of investigation. On a closer inspection, however, the apparently irregular exceptions are found more and more to concur; and finally it comes out as a constant and regular occurrence that under certain conditions which depend upon the place of excitation and on the condition of the eyes at the time of excitation, no associated eye-movements take place. If one stimulates the middle of the spot A (figs. 1 and 2), say of the left visual area, both eyes remain quite immovable if the dog is looking straight forward, or the left eye goes somewhat to the right whilst the right eye turns a little to the right or left; while if the dog is *not* looking straight forward the left eye always moves strongly to the right, whilst the right eye continues in this case also at rest, or only turns a *little* to the right or left. Now, as the upper eyelid is raised in all cases, and the pupil undergoes a sudden extensive dilatation, the desired certainty is obtainable that in the case of rest, or only slight movement of the eyes, the excitation was as effectual as before. If the excitation be brought nearer to the antero-lateral end of the visual area

by placing the electrodes upon the second convolution external to the fissure which divides it, or both there, and upon the third convolution—then both eyes move to the right, and at the same time more or less downwards, if the dog is looking fixedly forwards; but they converge considerably if it does not.

We have obtained more experimental results of this kind, but their conditions cannot so far be formulated with the same certainty. For the enquiry is rendered difficult by the fact that many dogs, after they are awakened from the ether narcosis, to which they are subjected for the operation, remain in constant excitement, and look continually first upon this, then upon that object, which may happen to be before their eyes. The only dogs which are perfectly serviceable for this experiment are those which are naturally patient or become quiet after the experiment has lasted a short time, so that they can be tested in a condition alternately of increased or diminished attentiveness, according to the degree of attention which is being devoted to them. In such dogs it is easy to make the observations, and they suffice to get rid of the objection now under consideration. This could not be done before, because with regard to the associated movements of the eyes, right, left, upwards or downwards, which Mr. Schaefer found in the ape, and which we have above shown to occur in the dog, there is nothing to distinguish them from common reflex movements. But the new investigations combined with the old ones, have led the results of stimulation beyond the limits of such movements. That the eyes by one and the same stimulation, at one time remain at rest, and at the other move; at one time make associated, at another converging movements, is not conceivable, if it is dependent upon excitation of nerve fibres or ordinary nerve cells. It is only comprehensible and agrees with what is to be expected if sensory perceptions are answerable for the result, if light perceptions cause the fixation of the eyes.

Other experimental results are confirmatory of these. If the electrodes, a weak excitation only being employed,

are shifted from the middle of spot A forwards and backwards (best when keeping to the second convolution on the mesial side of the fissure which divides it), the eyes move downwards the further forward one excites the area, and upwards the further backwards, and without exception the strongest downward movement that can be obtained is less than the strongest upward movement. In order to explain all this, without assuming visual perception, one must take refuge in a number of gratuitous and improbable suppositions. On the contrary, it can be easily and perfectly understood on the supposition of eye-movements which lead to the fixation of an object previously indistinctly seen, since the spot of most distinct vision in the dog is situated in the upper outer quadrant of the retina, where the tapetum is most developed. One can be in no doubt about the determination of this; and that it is right, is confirmed by further experiments. It is found, for example, in many dogs, that the strongest downward movement is not only less, but considerably less, than the strongest upward movement; and as the post-mortem section proves, there are always dogs whose tapetum, of normal size, is placed unusually high. In such dogs, the lower border of the tapetum runs at a considerable distance from the horizontal meridian of the optic papilla, while, in others, it reaches the horizontal meridian; occasionally even extending below it. Since obtaining this insight into the conditions of the retina and occipital lobe, we have been able to predicate regularly the position of the tapetum by the results of stimulation, the degree of difference between the strongest downward and the strongest upward movement being inseparably connected with the higher or lower position of the spot of distinct vision.

In the way thus marked out, by means, that is to say, of the results of electrical stimulation of the visual area, evidence of projection is obtained, which is entirely independent of the former evidence which I have given. From a scientific point of view the higher importance is undoubtedly to be attached to the earlier evidence, because the method which was employed, viz., that of extirpation,

leads directly to the point desired; whereas by the method of excitation this is only to be attained by circuitous routes, and not without the employment of intermediate links, if indeed these are altogether reliable. On the other hand, the new evidence offers this advantage that the easier and less involved experiments, in which stimulation combined with section is employed, would, judging from the preference hitherto evinced by investigators, be more likely to be repeated than extirpation experiments, which require months at least, and are difficult alike in execution and observation. At any rate, it is of unmistakeable value, that the conformity of the experiments by both methods warrants the assumption that there is some certainty in the knowledge acquired. And this conformity extends even farther than I have yet mentioned. For the result, which the stimulation of the antero-lateral extremity of the visual area offers, is only completely comprehensible, on the assumption that the antero-lateral part of the retina of the corresponding side is in connection with that extremity, and the results of excitation of the middle of area A are only rightly to be understood on the assumption that the centre of distinct vision of the opposite retina belongs to this area; and that the part indicated has been accurately hit upon in this latter stimulation, when both eyes remain at rest, or only one eye moves; but not quite accurately, if both eyes move. Mr. Schaefer's endeavour to rectify projection as discovered by me, so far as concerns the horizontal relations of visual area and retina, finds therefore its own condemnation by the facts which are elucidated by this method also.

As was natural to suppose would be the case, the recent exploration in a virgin district has, besides, led to a new acquisition. That the movements resulting from vision are brought about by impulses passing along two different paths, and that the lowest visual reflexes traverse the shortest path through the radiating fibres of the visual area, deepens our insight into the structure and functions, not only of the visual areas, but of the cerebral cortex in

THE VISUAL AREA OF THE CEREBRAL CORTEX, AND ITS RELATION TO EYE MOVEMENTS, BY PROFESSOR HERMANN MUNK (BERLIN).

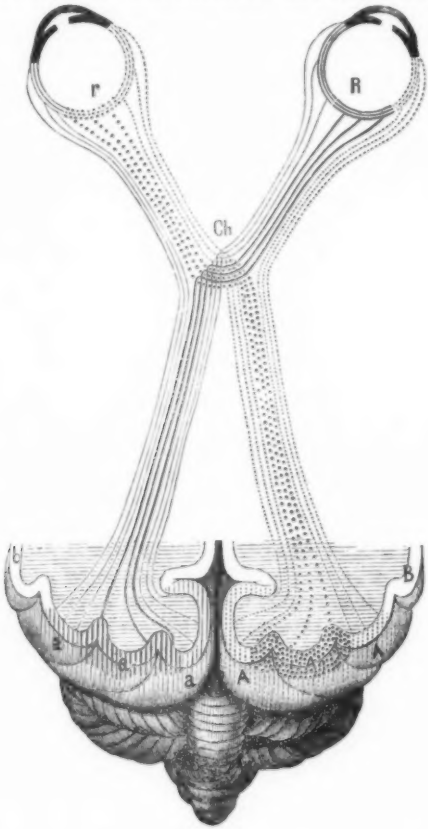

FIG. 1.

FIGS. 1 and 2 represent the connections of the visual areas with the retina in the dog (reproduced from Munk's "Uber die Functionen der Grosshirninde 5te Mittheilung.")

FIG. 1 represents a frontal section made through the middle of both visual areas. The eyes and optic tracts are represented cut through horizontally.

FIG. 2 represents both visual areas as seen from above. The right visual area A is dotted. The left (a) is marked with lines, and the spots (A_1 and a_1) the extirpation of which resulted in psychical blindness, are distinguished by darker shading. The posterior surfaces of both retinæ are shown, and the centre of each is marked C.

B and b indicate the auditory areas at their junction with the visual area. Ch.—Chiasma.

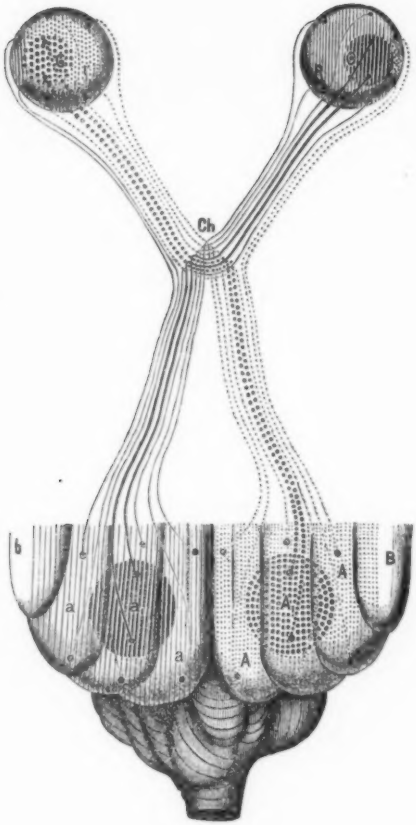

FIG. 2.

In both figures the portions of the cortex of the visual areas are connected by lines or dots with their respective corresponding portions of the retinæ.

FIG. 1 shows how each visual area is connected with the lateral fourth of the retina of the same side, and the mesial three-fourth of the opposite side, the most internal fibres of the retinæ passing to the most internal portion of the visual areas. The fibres coming from the central points of distinct vision are shaded darker and terminate in spots A and a.

FIG. 2.—The fibres may be traced from the upper borders of the retinæ to the anterior borders of the visual areas, from the lower borders of the retinæ to the posterior borders of the visual areas, and as in Fig. 1, the more deeply shaded fibres are seen passing from the centres of distinct vision to spots A and a.

With the exception of the outer portion, which is represented by lines in the left retina and by dots in the right, all the fibres proceeding from the retinæ cross over at the chiasma to the visual area of the opposite side.

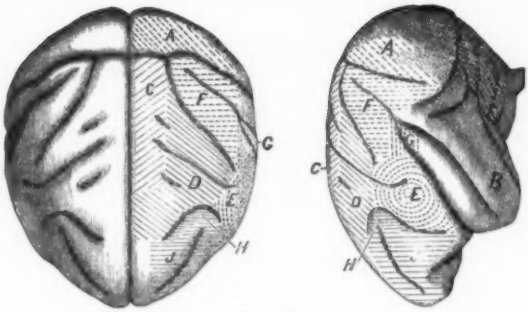

FIG. 3.

FIG. 3.—Cerebral cortex of the monkey (after Munk's "Functionen, &c., 4te Mittheilung.")

A.—Visual area.
C to J.—Tactile sensory areas—(Fühlsphaere) of Munk's, psychomotor areas of other authors.
D.—Fore limb region.
C.—Hind limb region.
E.—Head region.
F.—Eye region.
G.—Ear region.
H.—Neck region.
J.—Trunk region.
B is placed upon that portion of the cortex which from experiments on dogs is assumed to be the auditory area.

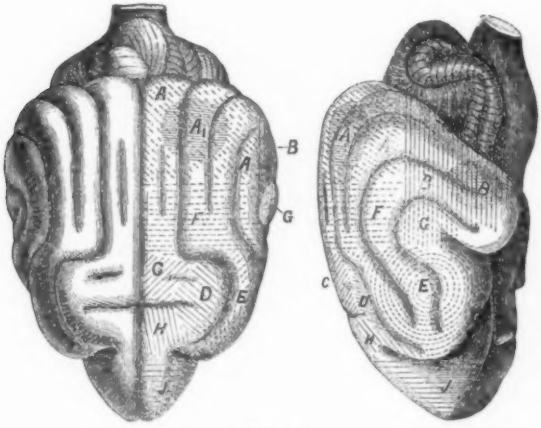

FIG. 4.

FIG. 4.—Cerebral cortex of the dog after Munk ("4te Mittheilung.")

A.—Visual area.
B.—Auditory area.
C to J.—Tactile sensory areas (Fühlsphaere).
D.—Fore-limb region.
C.—Hind-limb region.
E.—Head region.
F.—Eye region.
G.—Ear region.
H.—Neck region.
J.—Trunk region.

general, as will be subsequently shown. The projection of the retinæ upon the visual areas presents itself now in its full significance as the substratum for the localisation of the visual perceptions, since the involuntary eye-movements, which are brought about through the radiating fibres supply the necessary complement. Successive and opposite positions of the objects in Helmholtz's visual fields are yielded by projections, the judgment being assisted by the sensations which bring about these involuntary eye-movements, upwards, downwards, right, or left: thus projection and eye-movements together, permit such rapid and certain cognizance of the visual field, as we observe in animals, and which would be quite impossible, if it were necessary to deliberate regarding every detail in the visual field.

The discovery of the new radial fibres of the visual area will prevent the anatomical investigator from being able, without further consideration, to refer all descending degenerations which result from removal of that area to the tracts which conduct visual impressions, but there is on the other hand now opened up to him the prospect of being able to distinguish the two kinds of central elements, and of being able to demonstrate their morphological differences, a prospect which is attractive by reason of the proved connection of the radiating fibres with the light-perceiving central elements, and of the associated fibres with the representative elements (Vorstellungselementen).

Moreover, it is worthy of remark that our result, that the light-perceiving central elements are thrown into excitation by the electrical stimulation of the visual area, is not isolated. That the movements which result from electrical excitation of the psycho-motor area (Fuehlsphaere) proceed from the excitation of the grey cortex or cortical elements, has already been settled after much labour and research:[17] and is proved by the fact that if the electrodes lie on the uninjured surface it requires a weaker current to obtain a result, than if by the removal of the cortex the exposed white substance is stimulated; further, because the movements in the former case commence later, and last longer

than in the latter case, and lastly because, in dogs which are narcotised strongly with choral or morphia the movements disappear on excitation of the surface, not on excitation of the white substance. Corresponding statements apply also to the visual area. Here also it is found that after removal of the cortex, stronger currents are required to obtain the associated eye-movements,[18] than if the electrodes are placed on the uninjured surface, and it may also be shown that in dogs which are strongly narcotised by morphia or weakened by loss of blood, the excitation of the surface is unsuccessful unless a very strong current be used, whilst the excitation of the exposed white substance produces results with a current of ordinary strength. Hence it follows that the movements resulting from electrical stimulation of the visual area are derived from the excitation of the area itself, *i.e.*, of its cortical elements. One would, it must be freely admitted, need strong evidence to prove that only certain central elements of the cortex are thrown into excitation by the stimulation; evidence stronger than is as yet afforded even by all these considerations. One might, since the nerve fibres of the corona radiata are prolonged into the grey cortex, endeavour to explain the different results got from stimulation of the cortex and of the white substance, by considering that these nerve fibres have a different structure and irritability in the cortex and white substance respectively. Such a supposition is, however, constrained and unnatural, for an undoubted and great difference exists between the ganglion elements on the one hand, and the fibres on the other hand, and it is easily conceivable that the ganglion elements are excited by weaker currents, and are more easily injured by narcotics or insufficient blood supply than the nerve fibres. One is, therefore, justified in concluding from the foregoing considerations that the central elements of the visual area are excited by electrical stimulation of the same, and hence it follows that, as our above described research has proved in another way,[19] the excitation of the light-perceiving central elements through the electrical stimulation of the visual area represents the next advance in our knowledge.

In conclusion, I must emphasise the harmony which has existed between my earlier inquiries by extirpation and my later ones by stimulation. It goes further than projection, which was the object of our researches. It was astonishing every time that we came upon that spot of the visual area upon stimulation of which, if the dog were looking fixedly forwards, both eyes remained at rest, and, if the dog were not in this condition of fixation, the opposite eye remained immovable, how accurately this spot was found to correspond to the middle of the stelle A, *i.e.*, to the middle of those parts of the visual areas, by extirpation of which I produced psychical blindness, and which I afterwards proved to belong to that part of the retina which is concerned in distinct vision. But it is the same in respect to the borders of the whole visual area, when one takes into consideration how easily for such determinations the methods both of extirpation and stimulation are saddled with possibilities of inaccuracy, each in its own way. According to the impressions which I have formed from the excitation experiments, I should be inclined to believe that the anterior margin of the visual area, at its mesial extremity, was situated a little farther forward along the first convolution than my diagrams show; this would be in concurrence with the fact that in those cases in which the intended total extirpation of the visual area had not been perfectly successful, I had to seek for the remainder of the visual area with special frequency in the anterior mesial end of the area. On the other hand, the somewhat triangular corner which, according to my diagrams, is cut off at the antero-lateral border of the visual area from the third convolution, has now to be excluded from the visual area; manifestly, however, it is necessary for total extirpation of the visual areas to take away this triangular corner bit, in order that none of the cortex of the second convolution be left behind in the fissure between this and the third convolution.

LIST OF REFERENCES.

[1] Ueber die Functionen der Grosshirnrinde. *Gesammelte Mittheilungen.* Berlin, 1881. S. 31-4; 38.

[2] Ibid, S. 79-94.

[3] Ibid, S. 104.

[4] Ibid, S. 38, 126-7. Du Bois-Reymond's *Archiv.*, 1881. S. 457-9.

[5] *Philos. Transact. of the R. Soc. of London*, vol. clxxix. (1888), B. p. 315-7; 321-3. BRAIN; *a Journal of Neurology*, vol. x. (1888), p. 370-1: vol. xi., p. 1.

[6] BRAIN, vol. xi., p. 1-6. Everything of importance is here quoted word for word.

[7] In order to make this intelligible, I must remark that Schaefer's "visual area" extends, not only over the occipital lobes, but also over the immediate neighbourhood forwards and outwards. This is of no importance as concerns projection, about which my present communication treats, therefore I shall not further refer to it in the text. But in the interest of the subject it is important in passing to throw some light upon this "important difference." In the *Phil. Transact.*, vol. clxxix. (1888) B., the conclusion to which Schaefer has arrived as the result of his and S. Brown's experiments appears as follows:—

"The two experiments, in which the ablation respectively of one or both occipital lobes was complete, are entirely confirmatory of the statements of Munk on this point, who asserts that complete and permanent blindness is produced by the removal of the occipital lobes alone without the implication of the angular gyri, and that removal of one occipital lobe causes complete and permanent hemianopsia;" (p. 321) and again, "these two experiments, namely, those in which the occipital lobe was totally removed, one unilaterally and two bilaterally, appear to afford a complete demonstration of the idea that in the cerebral perception of visual impressions, that lobe and that lobe only of the cerebral hemisphere of the monkey is concerned" (p. 322). The same position is also taken up in BRAIN (vol. x., p. 362-72). "When temporary hemiopia has resulted in consequence of extensive lesions of the temporal lobe and in one case of the angular gyrus, these transient symptoms may be ascribed rather to disturbances of the circulation in the occipital lobes and to the temporary loss of support which is afforded to that lobe, than to the fact that the visual area of the cortex spreads over from the occipital lobe into the adjoining parts of the brain. If this were the case in the monkey there should be always some remains of the visual perceptive faculty after removal of the occipital lobes alone, whereas both Munk and ourselves have found that there may be no trace left of such faculty. . . . . It is, however, also possible that fibres connected with the cortex of adjoining parts of the brain (and especially of the angular gyrus), may curve backwards into the occipital lobes, and thus become cut off along with that lobe. Various facts might be cited in support of this idea. . . but we are not at present in a position to come to a definite decision upon the subject." Now the communication from which the above text (BRAIN, vol. xi., p. 1-6), is quoted, commences in the following manner:—"The visual area of the cerebral cortex in the monkey, so far as it is determinable from the results of extirpation and of electrical excitation, comprises the whole of the occipital lobe. It perhaps includes a part or the whole of the angular gyrus, although the results of extirpation would appear to show that the part played by the latter convolution is comparatively small. Removal

of both occipital lobes produce total and permanent blindness, whereas destruction of the cortex of both angular gyri is not followed by any appreciable permanent defect of vision. But, as will presently appear, electrical excitation of the posterior limb of the angular gyrus produces movements of the eyes similar to those which are produced by excitation of the adjoining portion of the occipital lobe; it is probably correct, therefore, to include at least this portion of the angular gyrus in the visual area." To this a foot-note is added. "A similar result is obtained on stimulation of the upper end of the superior temporal gyrus and of the adjacent portion of the middle temporal. This may be due, as is usually supposed, to the calling up of subjective auditory sensations; but the possibility of this part belonging also to the visual area with which it is anatomically continuous must not be overlooked." Again, on p. 4 it is stated—"I have not obtained any effect from stimulating *with weak faradisation* the anterior limb of the angular gyrus, even when well marked results have been obtained with a strength of current barely perceptible to the tongue, from the parts which have just been considered." (It is not clear what is meant by "the parts just considered;" perhaps the cortex of the occipital lobe.)

I have given in full the material for an estimation of the "very important difference," and I need add nothing to it. Schaefer must certainly have been aware that the electrical currents, when the electrodes are placed just in front of the borders of the occipital lobe, are not absolutely confined to the point which is in contact with the poles, and therefore the surrender of his earlier and correct position is only to be explained by the lively dispute in which he became engaged with Ferrier, as a result of his upholding my experiments (see BRAIN, vol. v., p. 7. 145). But I do not think that, because Ferrier, as is now universally acknowledged, at one time made certain useless experiments, more energy should be wasted in refuting his errors.

[8] Ueber die Functionen u. s. w., S. 127.

[9] Du Bois-Reymond's *Archiv.*, 1881, S. 458-9.

[10] Ueber die Functionen u. s. w., S. 126-7. Du Bois-Reymond's *Archiv.* 1881, S. 456-7.

[11] Ueber die Functionen u. s. w., S. 62. Du Bois-Reymond's *Archiv.*, 1878, S. 552.

[12] The following facts concerning some similar experiments became known to us in the spring of 1889, only after we had carried out our research, and we must be content with noticing them without comment.

Schaefer (*International Journal of Anatomy and Physiology*, 1888, vol. v., part 4), removed first the one, then the other frontal lobe in front of the fissure of Rolando, cutting through the corpus callosum as well; electrical stimulation of the occipital lobe then caused associated eye-movements towards the side opposite to the excitation. "It is therefore clear," says Schaefer, "that these posterior excitable regions do not *necessarily* produce the movement in question by calling into activity the grey matter of the frontal cortex, and the centre through which they operate after removal of that grey matter, must be looked for in some more deeply seated portion of the brain (very probably in the grey matter of the corpora quadrigemina). At the same time there is no evidence that when the frontal cortex is intact they do not operate through that centre," that is to say, as appears from the context, through the anterior motor centre in the frontal cortex. Further, Danillo has stated in a preliminary communication (which is, in many respects, not very clear since we have been dependent

upon a translation from the Russian) that the associated movements of the eyes to the opposite side which he produced by electrical stimulation of the white substance of the occipital lobe in very young dogs and cats, still remain after the removal of the cortex of the anterior motor region, likewise when he separated the anterior part of the brain from the posterior by a transverse section 1½ cm. deep, and the same even when he made deep longitudinal sections parallel to the mesial border of the hemisphere along the first occipital convolution and also in the region of the gyrus angularis. Danillo therefore concludes that the centres for associated eye-movements are neither in the motor nor in the occipital region of the cerebral cortex, but are deeply situated. Bechterew disputes this conclusion (*Neurologisches Centralbl.*, 1889, No. 18, p. 518, Note.) According to him such centres exist both in the motor and in the occipital regions. Their presence in the occipital lobes can, he believes, be considered as proved by this—that after sections which divide the cortex corresponding to the position of this centre from the deeper lying parts, their stimulation no longer produces the customary movements, and for this he cites his "Physiology of the Motor Area of the Cortex" in the Russian *Archives of Psychiatry*, 1886 and 1887, a communication about which no mention is elsewhere to be found.

Lastly, P. Rosenbach (*Neurol. Centralbl.*, 1889, No. 9, p. 255) has stated that "the lateral deviation of the eyeball, which is obtained with certainty from the occipital lobes, and best from a particular point in Munk's visual sphere, in all cases remains even after complete destruction of the motor region."

[13] *Diese Berichte*, 1889, S. 616.

[14] Compare Wernicke, *Lehrbuch der Gehirnkrankheiten*, Bd. i., S. 87, fig. 45*s*.

[15] *Diese Berichte*, 1889, S. 616.

[16] Ibid.

[17] Franck et Pitres, *Travaux du laboratoire* de M. Marey, ann. 1878-9. Paris 1880, p. 427-29; *Arch. de Physiologie*, 3. sér. t. v. 1885, p. 7. Bubnoff und Heidenhain, Pflüger's *Arch.*, Bd. 26, 1881, S. 140-70. François-Franck. *Leçons sur les fonctions motrices du cerveau*. Paris 1887, p. 29, 35, 318.

[18] The occurrence, on electrical excitation of the freely exposed white substance of the occipital lobe, of associated eye-movements towards the side opposite to the excitation has already been demonstrated by Danillo and P. Rosenbach (see Note 12). Danillo has observed the movements in very young cats and dogs, hardly two months old, in which excitation of the uninjured occipital lobes was quite unsuccessful, and he has concluded that therefore Ferrier's assumption that the eye-movements which are the consequence of cortical excitation depend upon subjective visual sensations, is untenable. That Danillo's conclusion is however, incorrect, now needs no further explanation, since the radiating fibres of the visual area, which conduct the excitation towards the periphery, are affected by the stimulation of the white substance.

[19] *Ueber die Functionen*, u. s. w., S. 89-91; 107-8.

A CONTRIBUTION TO THE PATHOLOGICAL ANATOMY OF CHOREA WITH THE REPORT OF A CASE.

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THE present paper deals chiefly with the pathological anatomy of the nervous centres in chorea. The changes elsewhere, particularly those in the heart have been often described, and there is a pretty definite agreement that the most uniform condition in patients dying of chorea or with chorea is fibrinous deposits on the valves of the heart, usually the mitral. Osler thinks that this occurs in 90 per cent. of cases.

I shall not take up this particular point, but turning to the nervous side of the question, I will give first the report of my own case.

Wm. G., Swiss, single, aged eighteen. Family history unknown. Admitted December 15th, 1888. Patient was always weak and sickly as a child. Has no clear remembrance of having any of the ordinary children's diseases; gives no rheumatic, venereal or alcoholic history.

Patient began to have choreic movements twelve years ago, *i.e.*, at the age of six years; cause unknown. The movements were not severe and when at the age of twelve he came to this country he was able to work and be useful.

The patient has had so-called epileptic attacks at occasional intervals for many years. The attacks used to occur at night only, but of late they have come on during the day also.

Physical Examination.—The patient lies in bed and is unable to walk or help himself on account of the violence of the choreic movements. He is somewhat emaciated and anæmic. His face is dull and the expression stupid, but he answers questions intelligently. The speech is slow and jerky on account of the move-

ments of the lips and tongue. Over the left parietal bone is a hole the size of a half-dollar made by a trephine in an operation performed in San Francisco fourteen months ago. No report of any hemiplegia or other form of paralysis. There are no heart murmurs, lungs normal, as are also the abdominal viscera. Urine yellow, clear, neutral, sp. gr. 1021, no albumin; microscopical examination negative. Patient's movements are so great that he cannot feed or help himself. He has several epileptoid attacks daily and nightly with tonic, and clonic and also with co-ordinate movements, throwing himself about, screaming, and frothing at the mouth. The fits are of a hysteroid character. Ordered chloral hydrate and frequent feeding with stimulants.

December 20th, five days later, hysteroid attacks are less numerous and only at night; choreic movements less, so that patient can feed himself. He is stupid from the chloral. Temp. $100\frac{1}{5}$, pulse 80, res. 20.

December 28th.—Thirteen days after admission, is much brighter, sits up, talks and reads quite well; chorea better. Ordered ext. physostigma gr. $\frac{1}{4}$. Diminished chloral. Patient improved considerably, the fits ceasing altogether, until January 6th, 1889, when he developed pneumonia and died January 8th.

Autopsy.—Rigor mortis well-marked, body considerably emaciated, left lung shows beginning pneumonia, heart normal; no vegitations on valves, liver and spleen normal, kidneys were small, capsule adherent and thickened; markings fairly plain, skull was extremely thick, measuring 14 ctm. ($\frac{7}{8}$ in.) on section through occipital bone. Brachycephalic in shape. Antero-post diameter 18, transverse diameter 16 ctm., giving a cephalic index of 88[1].

The old trephining hole lay over the left supra-marginal gyrus. The dura mater was not very adherent, the pia mater was thickened and congested, but not markedly adherent to the cortex. There was no excess of cerebro-spinal fluid. The brain looked normal, except for a very superficial softening seen on the under surface of both temporal lobes. The cord was apparently normal; the brain, cord, pieces of sciatic, musculo-spiral nerve and biceps were saved for further examination.

Microscopical Examination.

The brain, cord, and nerves were placed in Müller's fluid, frequently changed, and at the end of three months (for cord,

[1] I am indebted to my house-physician, Dr. Alex. Lambert, for the above carefully taken notes of this case.

bulb and nerves), and five months (for brain) placed in alcohol. Pieces of the motor convolutions, temporal, and parietal convolutions, as well as sections of the corpus striatum and optic thalamus were cut and stained. Sections through the pons, bulb and cord were also made. The stains used were Weigert's, the ordinary hæmatoxylin, carmine, and Congo red and Golgi's nitrate of silver. By the latter method very beautiful sections were obtained.

Convolutions and subcortical White Matter.—There is a slight amount of lepto-meningitis, the gray matter is considerably injected, the neuroglia cells are not excessively increased. The pyramidal cells are for the most part normal. Occasionally one finds a cell undergoing pigmentary degeneration. The outlines and processes and lymph paths come out beautifully by Golgi's stain. The nerve fibres can be seen running up into the cortex; the transverse fibres are also present. The chief changes are just beneath the cortex. Here the white matter is in some sections honeycombed with little spaces, round, oval, or like long slits. The spaces are either empty or partly filled by blood vessels (which latter are shrunken by the bichromate). The blood-vessel walls are somewhat thickened, but not strikingly so. There is no real arteritis. They have, however, been tensely distended, and exudates, including red blood cells poured out. This has distended and eroded the perivascular spaces, forming large dilatations, similar to those described by Dickenson. (See illustration.)

The process, though chronic, cannot be looked upon as inflammatory, and it can only be explained by assuming a flabbiness and loss of tonus of the vessel walls (due to prolonged distension) and allowing abnormal dilatation and filtration of the vessels' contents. There is no hyperplasia of connective tissue shown by Weigert or logwood stains, nor any amyloid bodies. At the base and tip of the temporal lobe, where the surface of the brain was softened a severer grade of meningitis or chronic meningeal thickness was present and the superficial layers of the cortex showed evidences of disintegration; the ganglion cells could be seen, but the white matter beneath was more extensively honeycombed and slit, while there was evidently increase of neuroglia cells.

Basal Ganglia and Internal Capsule.—These parts did not appear to the naked eye so much involved as did the subcortical tissue. However, the middle portions did not harden well, and only the optic thalamus, the lower part of the internal capsule and the anterior part of the caudate nucleus could be examined

microscopically. These parts showed the same honeycombed appearance from vascular dilatation, though less marked than in sections higher up. Here also it could be seen that the small arterioles formed varicose dilatations, which were full of blood. The internal capsule showed microscopically the more serious disorganisation. Its fibres were split up by interlacing and dilated vessels, whose walls were thin, degenerated and often seemed actually falling to pieces. Here I noticed, first, a varicosity of the nerve-fibres, caused by bulging of the myeline sheath, as described by Berkley. Cross sections of the capsule stained badly with Weigert, and seemed to be full of fibres swollen or partly disorganised. There was also a decided hyperplasia of connective tissue in the capsule, which was not noted in the thalamus or caudate nucleus.[1]

The corpora quadrigemina were dotted and streaked with dilated capillaries, but showed no large perivascular spaces.

Crura Cerebri and III. *Nerve Level.*—There is a less degree of vascularity than at higher levels; cells of III. N. nucleus are normal, so also are the fibres of the crus and the cells of the substantia nigra.

Descending root of V. N. normal.

Pons Varolii.—The nucleus of the IV. N. contains few cells and some of these seem degenerated. The fourth nerves appear to have more degenerated fibres. The cells of the motor nucleus of the V. seemed to be normal in size and number, without pigmentation or vacuolation. The blood vessels are numerous and dilated, and their walls are thicker than those seen in the brain.

Upper Medulla.—A neuro-fibroma, the size of a pea, is seen just at the exit of the acoustic nerve on the left side, the fibres of the acoustic pass around it, a few passing into it. The only nerve nuclei in which I could detect signs of degeneration were those of the vagus (including the anterior nucleus) and accessories. The gray matter of the floor of the fourth ventricle was much honeycombed with very small cavities made by dilated arteries. There was apparently some increase of connective tissue in the pyramidal tracts. The neuro-fibroma referred to had not destroyed any parts by pressure.

Spinal Cord.—The pia mater was thickened, somewhat adherent and filled with distended vessels. The spinal cord showed some congestion, the distended and thickened vessels being most

[1] I could find no hyaline bodies such as are described by Dickenson and others.

numerous in the lateral columns. There was none of the honeycombed appearance as found in the brain and pons. In the lumbar cord a double canal was present, and for a short distance a third canal; a slight increase of interstitial tissue was observed in the lateral columns. The anteror and posterior roots showed no degenerated fibres.

The Nerves.—Sections of the anterior tibial nerve showed no inflammatory or degenerative changes whatever.

SUMMARY.—Brain, chronic leptomeningitis, non-adhesive, and therefore not severe. Diffuse and varicose dilatations of the small arteries, especially of the deeper sub-cortical matter and capsule. Degenerative changes in arterial walls; no arteritis. Perivascular lymph-spaces greatly dilated. Cortical cells in most regions normal. The severest changes, vascular, interstitial and degenerative were in the under surface of the temporal lobes in the internal capsule and adjacent parts of corpus striatum (especially lenticular nucleus), and optic thalamus (antero-internal part). Varicose nerve fibres were here noticed.

Pons Varolii and Medulla.—Same condition but much less marked. Cell degeneration in some cranial nerve nuclei. Slight connective tissue increase in pyramidal tracts.

Spinal Cord.—Slight lepto-meningitis, congestion of cord, especially in lateral tracts. Double central canal.

The record of the foregoing case naturally leads one to turn to previous accounts of autopsies for comparison. We here notice, first of all a curious distribution of these records.

The mortality from chorea seems to be much greater in England than anywhere else. In no other country have so many autopsies been made.[1] Of the English hospital cases 3 or 4 per cent. die. The Collective Investigation Committee found a mortality of over 2 per cent from chorea. Some years ago French physicians saw a good deal of fatal chorea, but few reports are made by them now. In Germany, there have been very few contributions to the pathological anatomy of chorea. In America I know of only three cases reported in detail.

The English observers have almost always examined acute cases, and their findings chiefly pertain to the vascular system; subinflammatory states, exudations, embolisms, &c.,

[1] Stuges collects eighty fatal cases reported by six English hospital physicans.

being noted. The Germans have seen more in the connective and nerve tissues, and note hyperplasia of the former with degenerative changes of the ganglion cells.

The French have as yet contributed few autopsical records of positive value.

I have examined and tabulated the *post-mortem* records in over eighty cases.

Raymond collected and tabulated (*Dict. Encyclopæd. de Sc. Med.*) seventy-nine cases. Seé in 1850 (*Memorie de l'Acad. de Med.*) collected eighty-four reports of autopsies. Eliminating the duplicated cases, I find that there are on record nearly 200 reports of autopsies made upon patients dying of, or with acute or chronic chorea.

The large majority of these are of no value whatever, so far as the observations on the nervous system are concerned. In some in which careful examinations were made, the chorea was accompanied with mania, as in Golgi's case, or was symptomatic as in Froriep's case, or was of the hereditary type, as in Macleod's.

From a critical analysis of the list of reports, I can find only thirty-nine cases which give a satisfactory account of the nervous system. Even here, in some instances, the examination is not perfectly complete.

The list of valuable records, so far as I can find them, is as follows:—

1. Rokitansky, *Sitz. ber. der. Wien. Akad. der. Wiss*, 1837.

2. Steiner, *Prag. Vierteljahrsch.*, 1868, 25 Bd. iii.

3. Meynert, *Wien. Med. Presse*, 1868, pp. 194.

4. Oppolzer, cited by Rosenthal; *Wien. Med. Presse*, 1868, p. 195.

5. Tuckwell, "St. Barth. Hosp. Rept.," vol. v., p. 86.

6. Russell, *Med. Times and Gazette*, 1865, p. 88.

7. E. L. Fox and R. S. Smith, *Med. Times and Gazette*, 1870, p. 423.

8. Aitken, *Glasgow Med. Journal*, 1853, vol. i., p. 92.

9. L. Clarke.

10. Naunym, Schmidt's *Jahrbüch*, Jan. 15, 1889.

11. Elischer, *Virch. Archiv*, vols. lxi., p. 485 and lxiii., p. 104.

12 to 18. DICKENSON, *Medico-Chirurg. Trans.*, 1876, vol. xli.

19. J. H. HUTCHINSON, *Phil. Med. Times*, 1876.

20. DONKIN and HEBB, *Med. Times and Gazette*, 1884, Nov. 1.

21. GOLGI, *Rivista Clinica*, 1874, p. 361.

22. BERKLEY, *Medical News*, Aug. 25, 1883.

23. ROSS, "Diseases of Nervous System," vol. ii.

24. BURY, quoted by Ross (*loc. cit.*)

25. KELLY, *Trans. Path. Society of London*, vol. xxiii., p. 94.

26. CL. DE BOYER, *Bull. Societ. Anat.*, 1875, t. xx., p. 551.

27. M. GUINON, *La France Medicale*, Jan. 19, 1886.

28. F. ROLAND, *La Progres Medicale*, 1886, vol. iv. p. 893.

29. HANDFORD and POWELL, "Brain," July, 1889.

30. Ditto ditto ditto.

31. DANA.

32. PATELLA, *Gazz. degli Ospital*, Sept., 1888.

33 to 39. JAKOWENKO, *Wiestnik psichiatrii i. nervopatol*, 1889, ii.

In excluding from my table so many records made, in many instances by careful observers, I do not mean to deny their value. These observations have shown in a general way that in death from chorea, congestions, extravasations, embolisms, and softenings, exist in the nervous centres. They have also proved the great uniformity of heart lesions. It is, however, only by a very minute and careful microscopical study of cases that any light upon the neuro-pathology of chorea can be obtained. For it does not follow because the cord is often found soft, or the walls of the central ventricles pultaceous, or that clots are found in the central arteries in fatal chorea that any such change occurs in the ordinary type of the disease. And the large number of incomplete autopsies upon chorea has simply confused our notions of it.

This confusion as to the pathological anatomy of chorea has been increased by the fact that systematic writers

No.	Reporter.	No. of Cases.	Age.	Sex	Cause.	Duration.	Type & Complications.	Findings.
1	ROKITANSKY ...	.....	...	...	.....	.....	....	Increase of connective tissue in brain and cord.
2	STEINER	3	...	...	.....	.....	.....	Increase of connective tissue in spinal cord, with serous exudation and effusion of blood in spinal canal.
3	MEYNERT ...	1	16	F.	.....	.....	Chorea of Sydenham.	Congestion and sclerotic changes in brain, colloid bodies. Degenerative changes in cells of spinal cord.
4	OPPOLZER ...	.....	...	M.	.....	.....	.....	Increase of connective tissue in cord, softening in its upper dorsal and lower lumbar parts.
5	TUCKWELL ...	.....	13	F.	Preceded by rheumatism.	3 weeks.	Chorea of Syd.	Emboli in both posterior cerebral arteries and partial softening in under surface of temporal lobes. Spinal cord normal.
6	RUSSELL	....	14	M.	Rheumatic.	About 1 month.	Chorea of Syd.	Brain atrophy, increased subarachnoid fluid. Pial ecchymones. Brain vessels empty, cord and corpus striatum examined only.
7	E. L. FOX ...	....	17	F.	Fright.	1 month.	Chorea of Syd.	Meningeal hæmorrhage, small vessels of corpus striatum engorged with blood. Other parts not examined.
8	AITKEN	.....	17	F.	.....	10 days.	Sub-acute chorea, imbecility.	Varicose dilatation of blood vessels of gray matter of brain. Lessened spec.-grav. of basal ganglia.
9	L. CLARKE							
10	NAUNYM	.....	17	F.	.....	6 years.	Severe type of chronic chorea.	Oedema of brain and cord. Brown-red coloration of pia in fissure of sylvius and at base, caused by fungi growing along the vessels.
11	ELISCHER ...	.....	22	F.	3rd attack. ...	4 months.	Chorea of pregnancy.	Diffuse irritative process causing increased proliferation of nuclei of neuroglia and of vessel walls. Signs of degenerative process in nerve cells.
12	DICKENSON ...	8	10	F.	Rheumatic. ...	3 days.	Chorea of Syd.	Hyperæmia of parts of brain and especially of cord. Hydromyelia, especially of lower cord (congenital?) Hæmatomyelia of upper part of cord.
13	Ibid ...	2	10	F.	Fright, 3rd attack.	3 weeks.	Chorea of Syd.	Distension of arteries and veins and effusion in perivascular spaces. Congestion of cord.
14	Ibid ...	3	7	F.	?	24 days.	Chorea of Syd.	Distension of arteries and veins, especially of optic thalamus, congestion and extravasation in cord.
15	Ibid ...	4	8	F.	Fright.	2 months.	Chorea of Syd.	Congestion of brain and cord.
16	Ibid ...	5	11	M.	Rheumatism.	6 weeks.	Chorea of Syd.	Congestion of corp. striatam and in the white matter of one of the convolutions with signs of small embolisms? Congestion and sclerosis of gray matter of cord.
17	Ibid ...	6	13	F.	Rheumatic, 3rd attack.	3 weeks.	Chorea of Syd.	Congestion of basal parts of brain and old spots of sclerosis? especially in motor convolutions. Congestion of cord.
18	Ibid ...	7	54	M.	?	4 years.	Chronic chorea.	Congestion of brain with old spots of so-called sclerosis (artefacts?) especially in basal parts and corpora striata.

					Chronic chorea.		Congestion of brain with old spots of so-called sclerosis (artefacts?) especially in basal parts and corpora striata.	Congestion of cord.

No.	Reporter.	No. of Cases.	Age.	Sex.	Cause.	Duration.	Type & Complications.	Findings.
19	J. H. Hutchinson	1	12	M.	Fall.	3 weeks.	Chorea of Syd.	Congestion of brain with some plugging? of arteries of corpora striata, cord is soft, 3rd to 5th dorsal.
20	Donkin & Hebb	.....	20	F.	Fright.	7 days.	Chorea of Syd. (?) with mania.	Brain very soft, walls of vessels disintegrated, cord showed congenital abnormalities, blood vessels full of micro-organisms.
21	Golgi	.....	42	M.	.....	10 years.	Chronic symptomatic, with mania.	Pachy and leptomeningitis, atrophy of cerebral convolutions white matter hyperaemic, spots of softening and gray degeneration, corpora striata softened, cord soft, membranes thick, ganglion cells atrophied, varicose axis cylinders, hyperplasia of connective tissue in cord and cell pigmentation.
22	Berkley... ...	.....	41	F.	Fright.	7 years.	Chronic chorea, imbecility.	Dilatation and thickening of arterioles with necrotic areas about them, degenerative changes in ganglion cells, varicosities of nerve fibres, amyloid bodies. Same changes very much less marked in bulb. Same changes quite marked together with sclerotic changes in cord, no embolisms.
23	Ross...	.....	...	...	.....	.....	Chorea of Syd.	Hyperaemia of spinal cord, especially of anterior and antero-lateral arteries. Degeneration of accessory nerve cells.
24	Bury	.....	...	...	.....	.....	Chorea of Syd.	Periarterial exudations and erosions; and lesions in general like those described by Dickenson.
25	Kelly	.....	9	F.	?	15 days.	Chorea of Syd.	Soft spots and small extravasations of blood in corp. striat. Fatty degeneration of capillaries and small arteries.
26	Cl. de Boyer	.....	7	F.	.....	4 months.	Chorea of Syd.	Marked meningo-encephalitis and cerebral atrophy, other parts normal. Microscopical examination (?)
27	Guinon	.....	19	F.	.....	2 months.	Chorea of Syd.	Nothing.
28	Roland	.....	7	F.	Scarlet fever.	Acute.	Chorea of Syd.	Very great cerebral hyperaemia, two pseudo cysts of the choroid plexus.
29	H. Handford & E. Powell	.....	19	M.	.....	21 days.	Chorea of Syd., with insanity.	Intense cerebral hyperaemia, thromboses and minute hæmorrhages in pons and upper cord.
30	H. Handford & E. Powell	.....	20	F.	.....	2 weeks.	Chorea of Syd., with insanity.	Same as No. 29.
31	Dana	.....	19	M.	.....	12 years.	Chronic with epilepsy.	Vascular dilations and perivascular erosions.
32	Patella... ...	.....	11	...	.....	.....	Chorea of Syd.	Softened areas subcortical, especially in occipital lobes. Hyperaemia, embolisms (?)
33 to 39	Jakowenko ...	6	...	...	.....	.....	Chorea minor. later delirium acutum.	Small hyaline bodies in globus pallidus lying about vessels, *vide* Meynert, Dickenson, Berkley.

have usually cited all the various findings in fatal cases as evidence of the lack of any definite anatomical basis of the disease. In fact the records in sub-acute cases, in chronic cases, in juvenile or adult cases, and in complicated cases, should be studied separately, and compared together with the facts of the clinical history kept in view. In this way, it will be found that there is very much less discrepancy in results than is supposed.

In the lists of fatal cases collected are :—

1. Sub-acute chorea of Sydenham.
2. Chronic ,, ,,
3. Hereditary ,, (Huntingdon's).
4. Congenital ,,
5. Symptomatic ,,

It is only the first two classes of cases that form the chorea as ordinarily met with. It is this disease whose pathology and anatomy is here specifically sought, and whose autopsical records I have collected.

I shall analyse my table with reference—

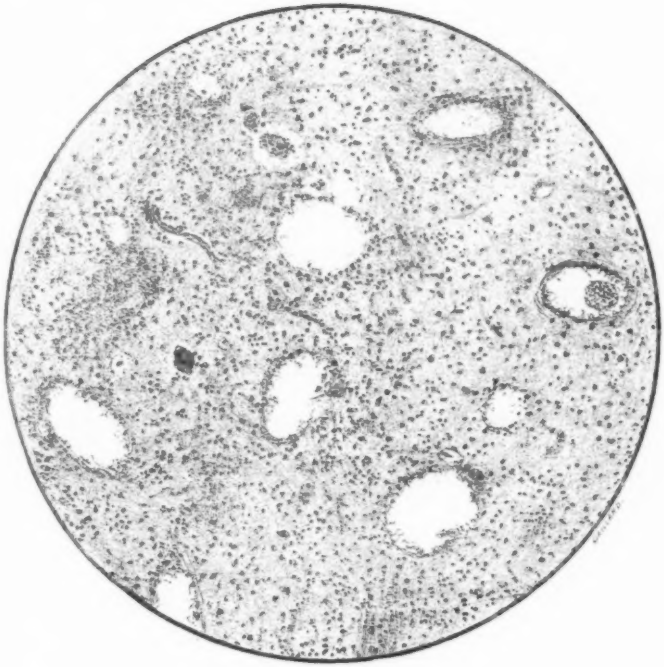
1st. To sub-acute chorea :

Among the thirty-nine cases there are twenty-five in which the disease was the sub-acute chorea of Sydenham, uncomplicated with insanity, or other serious disorder. Leaving out the six cases of Jakowenko, which are published in Russian, and not accessible in detail to me, there remain nineteen cases, in which careful microscopical examinations were made. In sixteen of these there is a record of intense cerebral hyperæmia, periarterial exudations, and erosions, softened spots and minute hæmorrhages, and occasionally embolisms. The changes are most marked, not in the meninges, but in the deeper parts of the motor tract, the lenticular nucleus and inner parts of the thalamus.

In a few of the cases, only, is the cord affected somewhat similarly.

Of the remaining three cases, in one (Roland's) absolutely no changes are recorded. I cannot but think that they were overlooked, or that a mistake was made in some way.

In one case, only the corpus striatum and cord were examined, and here spots of softening were found; and in one



case, the brain was very soft, the vessel walls were disintegrated, and full of micro-organisms.

In two cases of sub-acute chorea with insanity, there was intense cerebral hyperæmia with thrombosis and minute hæmorrhages, much as in uncomplicated cases.

In the six cases of Jakowenko, he says nothing about congestion, but describes minute hyaline bodies, found chiefly in the inner divisions of the lenticular nucleus (*globus pallidus*).

2nd. Chronic chorea.

There are records of five cases of chronic chorea, lasting from four to twelve years.

Here we find dilatation and thickening of arterioles with necrotic and erosive changes about them, degenerative changes in ganglion cells, varicosities of nerve fibres, old spots of sclerosis, and hyaline bodies.

The cord is somewhat similarly involved, but to a less extent.

There are no evidences of inflammation, or of continuous humoral irritations, such as eventually lead to decided arterial thickening.

Summing up we find that in the subacute types of chorea there is a hyperæmia of the brain and parts of the cord. In the brain this is not meningeal but subcortical and basal. The arterial walls are paralysed, dilated and badly nourished, so that exudations occur, and the lymph spaces become distended and eroded. There is sometimes stasis, thrombosis and spots of softening, or the walls give way, and there are minute hæmorrhages. The lymph spaces around the ganglion cells are not dilated.

In older cases, the vascular and neuro-degenerative changes are marked. The small arteries are permanently dilated, a little thickened and degenerated, perivascular channels may be more eroded and distended.

There is now some connective tissue proliferation, and signs of degeneration in the ganglionic cells. The nerve-fibres show a varicosity. (Berkley's case and my own). Hyaline bodies are seen.

In fine, we have in chorea, first a vaso-motor paralysis

and trophic disturbance, affecting certain areas of the brain, and to a less extent of the cord. Then we have this becoming chronic, with connective tissue, hyperplasia and degenerative changes in ganglionic cells and fibres.

As for the localisation of the lesion, the earlier English writers all looked for it in the basal ganglia, and their findings are, perhaps, a little biassed by the expectation of seeing something in these localities. In some autopsies only the cord and basal ganglia were examined. We cannot, however, locate chorea in these places exclusively, nor in the globus pallidus, nor in the cortex. It is rather a disease of the intracranial motor tract, including its starting point in the cortex, and especially in its co-ordinating adjuncts, the lenticular nucleus and thalamus.

NOTES ON A CASE OF TUMOUR OF THE CEREBELLUM WITH AN ABSENCE OF ALL SYMPTOMS.

BY P. WM. MACDONALD, M.D.

(Read at a meeting of the Dorset and West Hants Branch of the British Medical Association.)

In placing on record the following facts and notes my object is two-fold. Firstly, to show the value of negative facts and secondly, to prove the existence of a tumour in the left cerebellar hemisphere, without even a solitary symptom.

The respect due to old age may be said to reach the "zenith" of its depth and sincerity, when at the close of four score years and nine, a hitherto unknown mine of pathological wealth is discovered.

M. A. H. was admitted into the Dorset Asylum in December, 1864. From the Case Books and Registers, I learn, that her case was an ordinary one of maniacal excitement and mental perversion with many fixed and absurd delusions. During her residence in the asylum—which extended over a period of twenty-four years—her mental state drifted slowly towards the goal of dementia, in which condition it was for many of the latter years of her life. At the same time, though her mental state was one of dementia, she was conscious of all her surroundings and could answer questions till the last. One of her favourite themes of conversation was to make a few most affectionate enquiries after your health, and then, without any warning and before that you had time to move, she would pour forth in rapid succession, chains of the most obscene and disgusting language possible to imagine. Such are the vagaries of the human mind, which, I mean the mind, is ever an uncertain quantity, and of questionable quality when the higher or controlling bricks are from whatever cause disarranged, disorganised, or generally out of repair.

This poor woman was a member of the bronchitic family, and

often have I heard her say: "Doctor, cannot you cure the bronchitis?" Her appeals were not to be gratified, for on the 21st day of June last did this same bronchitis manifest its existence by an unexpected and grand display of renewed activity. No sooner had the fire been well ignited than the general grate began to fall to pieces, and seven days thereafter, or on the 28th day of June, did this same fine old specimen of eighty-nine human summers join the majority from natural causes, to wit, bronchitis. Into these few sentences I have squeezed a life record of four-and-twenty years.

My interest in the dear old woman did not lessen notwithstanding her disappearance from all that was mortal, and I respectfully followed her body to the mortuary. I will now tell you what we found there, or to be more precise, relate to you what careful dissections revealed.

In the thorax we found a comparatively healthy heart with a dilated aorta. The lungs were old offenders and everywhere we found the changes incident to long-continued bronchitis. Posteriorly and at the base the lung tissue was engorged and the larger and middle-sized bronchi were congested and contained thickish secretion. The abdominal organs, with the exception of the liver, were fairly healthy, but slightly congested. The liver was of an irregular shape, with rounded edges, pale, soft, and friable. On section hyper-glistening and a typical specimen of slow fatty changes. There was a small intra-mural uterine fibroid, and the gastro-intestinal tract was normal. We now proceeded to examine the head, and after removing an unsually soft and porous skull-cap, we came to a pale, thickened dura-mater with many adhesions to the cranium. The arachnoid was milky. No excess of subarachnoid fluid. Pia mater attenuated but no pia cortical adhesions. On cutting through the tentorium cerebelli on the left side we observed that the left hemisphere or lobe of the cerebellum was presssd upon by a round greyish coloured tumour, the size of a small apple, softish on the outer side, and adherent to the occipital bone. The surface of bony adhesion was the size of a shilling and the bone was simply a little rougher than natural. This was the only attachment to bone.

The brain removed it was noticed that the grey matter of the cerebrum was of an ashy-grey colour, while the central medulla was anæmic. Various ganglia normal. Arteries extensively diseased, being thickened and calcareous. The corpous callosum was softened. Taken as a whole the brain was wanting in consistence and volume. No special localised softening or lesion other than the cerebellar tumour.

Turning our attention to the tumour we note that it is a round encapsulated growth pressing on and causing atrophy of the left cerebellar lobe. In all probability it had its origin in the dura mater, and the attachment to the bone was of secondary occurrence and what we should expect to find.

In its development the main and principal increase of structure was towards the cerebellum, and it could not have had much tendency to grow in an outward direction, for its attachment to the occipital bone was only sufficient to produce an unnatural roughening on the surface of the internal table. Except at the point of origin where it is rather soft, the tumour is firm and compact, and from its general character will be placed among the sarcomatous tumours. Considering the size of the tumour, the small degree of displacement or destruction of the cerebellum is remarkable.

Microscopically the tumour is formed of small spindle nucleated and granular cells, and many numerous cellular and fibrillated elements. The somewhat rare appearance of the spindle cells being arranged in concentric nests was observed. The tumour then is a small spindle celled sarcoma.

So much for the characters and nature of the tumour, and next as to its presence and yet no symptoms. At no time during the patient's life in the asylum, nor before as far as I know was there any symptom of cerebellar disease.

We are told that extensive and even gross destruction of the lateral lobes of the cerebellum, may take place without producing any obvious symptom. The case under discussion must be placed among the unknown or undiagnosed but, though this has to be admitted, we may not unprofitably review and compare the material at our command with recorded facts and opinions.

By a process of slow and gradual pressure the tumour caused the destruction of at least one-third of the left hemisphere of the cerebellum. This depression in the cerebellum which was the result of atrophy (from pressure) proceeded in a central direction but not as far as the middle lobe, thereby leaving this the most important lobe of the cerebellum unaffected. The cases are few and rare where symptoms are absolutely wanting. In writing of this class of cases Dr. Byrom Bramwell says: "We must conclude then, that in some rare cases (in which an intracranial tumour is stationary or of extremely slow growth; in which there

is no increase of intracranial tension; and no irritation in the neighbourhood of the lesion) all symptoms may be entirely wanting." In the present case there were not even transitory symptoms to lead us to suspect an intracranial growth, and mainly for this reason the eyes were not examined. Though I can vouch for the absence of two (almost pathognomonic) symptoms, viz., headache and vomiting, I cannot definitely state that a third, viz., optic neuritis, was not present. The patient's sight was above or better than the average at 89. With the aid of cheap spectacles she could read small print. But this is no proof of the absence of optic neuritis, for it is now an accepted fact that optic neuritis may be associated with perfect central vision. Therefore it is to be regretted, that the eyes were not examined; at the same time, I am of the opinion that neither optic neuritis nor atrophy existed.

I do not think that a lesion in the lateral hemisphere of the cerebellum will *per se* cause any symptom by which it can be recognised, and the present case supports this view which is the opinion of our best authors on nervous diseases. Dr. Ferrier says: "the functions of the lobules of the lateral lobe of the cerebellum are principally concerned in various mixed movements of the eyes and head." This he has demonstrated by experiment and adds "it is only when the disease has been of a comparatively limited extent or confined to one lobe that there has been an absence of the characteristic symptoms."

Dr. Gowers says: "the hemispheres may be destroyed without recognisable symptoms, but this is not true of the middle lobe."

In the case under consideration we had actual loss of substance in the left lobe, but no interference with the functions of the middle lobe, hence no recognisable symptom. We can understand the presence of a small lesion and no symptoms, but when we meet with a large tumour—(such as I have shown you to day)—causing gross destruction, and still no symptom, we are constrained, to ask the question—are the accepted views and theories regarding the relations of the grey nuclei and peduncular fibres (in the lateral lobes of the cerebellum) to be relied upon or do they require revision and confirmation?

I have stated that at least one-third of the left hemisphere had undergone atrophy from the presence of the new growth, and as a result, we have the "semi-lunar tract" obliterated, and the outer margin of the "corpous dentatum" compressed, yet withal no symptoms.

From these facts we might build the hypothesis that the hemispheres of the cerebellum are mainly subservient to psychical processes and that a large lesion may exist without producing any recognisable symptom so long as the middle lobe is not compressed.

I invariably advocate the recording of negative facts, believing as I do, that such facts are often of as much value as positive ones, in elucidating and solving all the mysteries surrounding the functions of the greater and lesser brain.

The hypothesis that the cerebellum presides over the functions of organic life was we are told, founded on false anatomical views, and "there is indeed little or no evidence that the cerebellum is essential to the due performance of the functions of organic or vegetative life." (Dr. Ferrier).

"As we descend the scale the hemispheres of the cerebellum lessen, until they disappear in birds, in which the whole cerebellum corresponds to the middle lobe of man."

"Moreover the cerebellar hemispheres have this in common with the parts of the cerebral cortex with which they are connected, that simple loss of substance causes no definite and recognisable loss of any function of the brain." (Dr. Gowers).

Finally, "The effects of injury and disease show that the functions of the cerebellum are outside the sphere of mind proper, as expressed in sensation, emotion, volition and intellect." (Dr. Ferrier).

One word in conclusion. Was the tumour in this case the cause of the mental disease? No. I do not think it was the cause, but it may have been a secondary and irritative cause, and therefore, cannot wholly be raised from the platform of cause and effect. This view is the more tenable if we place any credence in the old theory that the cerebellum is in some way connected with psychical procesess.

October, 1889.

CASE OF HÆMORRHAGES IN AND ABOUT THE PONS.

BY J. S. RISIEN RUSSELL, M.B., AND JAMES TAYLOR, M.A, M.B.

We are indebted to Dr. Ramskill for permission to use the following case, which, on account of its rarity and the many points of interest connected with it, seems worthy of being placed permanently on record.

The patient dated the commencement of his illness from January, 1887, when he began to feel weak, to lose flesh, and to suffer from frequent attacks of frontal headache. He continued in the same somewhat indefinite condition for a year. At the end of that time he had a sudden loss of power in the right side, accompanied by sickness and vomiting, but without loss of consciousness. This passed off sufficiently in three days to permit of his walking about as usual, but from that time he remained weak. He had two attacks of giddiness subsequently—one two months later and the other in December last, without loss of consciousness or vomiting, but with a subsequent loss of ability to talk—in one case for four hours, and in the other for half an hour. There was no increase of weakness after either of those attacks. During the time he had been ill his memory became very defective, and from being an active, busy man, he became dull and apathetic.

When he came under observation at the National Hospital for Paralysis and Epilepsy, Queen Square, in April, 1889, he presented general weakness in all four limbs, more marked in the right arm and leg, and his gait was slow and somewhat tottering, but without any distinct hemiplegic character. There was no asymmetry of the face, and the tongue was protruded in the middle line. His utterance

was somewhat blurred. He understood what was said to him, and although he could not give a reliable history of his illness, apparently from impairment of memory, he was quite aware of his condition and surroundings. He was very drowsy, and it was difficult to fix his attention for more than a few minutes at a time.

A week after admission he was noticed to have lost almost entirely the power in his left arm and leg, while the limbs on the right side remained practically as before. Both sides of the face were affected, but the right much more than the left. The tongue was protruded to the left, and the articulation was more indistinct than formerly. He gradually after this became more and more drowsy and lethargic, and passed his evacuations in bed. He could still protrude his tongue, although he required a good deal of pressure to make him do so. He had considerable difficulty in swallowing, or rather a tendency to allow the food to remain in his mouth as if it were too much trouble to get it further. There was no change in the paralysis, but he gradually passed into a condition of coma, and died with a high temperature three weeks from the date of admission. He had no ocular paralysis and no optic neuritis, nor had he anything in the nature of a fit, except the three attacks already mentioned—one at the commencement of his illness and the others subsequently. There was slight albuminuria present, and evidence of a hypertrophied left ventricle.

It will be seen that this case presented some difficulties in diagnosis. Evidently there was some condition causing interruption of the tracts at some point between the cortex cerebri and the spinal cord, but there was difficulty in defining firstly, the site of the lesion, and secondly, its nature. An ordinary cerebral hæmorrhage first into one side of the brain and then into the other, was improbable, because in the first place there was no loss of consciousness at any time, and in the second, the recovery from the first attack was more rapid and probably more complete than is usually the case. Yet the presence of kidney disease and a hypertrophied left ventricle made it likely that the condition of arteries was that usually associated with cerebral hæmorrhage.

Tumour naturally suggested itself as a possible explanation of the symptoms, for these were ingravescent, and there was much headache and drowsiness. To cause the symptoms a tumour would require to be situated at a point where the tracts of opposite sides approach each other more or less closely. The localities in which the tracts are in such proximity as to be liable to pressure by a single growth are the medulla, the pons, the crura and the internal capsules. A tumour of the medulla such as to give rise to the condition of the extremities which was present here could hardly exist without producing symptoms of pressure on some cranial nerves, and the same might be said of the pons, where a growth of such extent as to cause the degree of paralysis here present would almost certainly give rise to oculo-motor paralysis.

Again, a tumour affecting both crura was excluded by the fact that a tumour large enough to cause the amount of paralysis present here would almost certainly have involved one, if not both, third nerves. Besides, as the face fibres in the crura are internal to those for the arm and leg, it is evident that a tumour pressing on both crura would first and most severely affect the face fibres, while the affection of the extremities would be secondary and less severe. The very opposite was the case here.

The only remaining point where the fibres of the motor tracts are in reasonable proximity is in the internal capsules, and a tumour pressing on both capsules seemed a feasible hypothesis to account for the symptoms. Dr. Bristowe in his recent volume on 'Diseases of the Nervous System,' relates five cases in which pressure was exerted at this point, in four of them by a tumour of the corpus callosum, and in the fifth by a growth starting in the lateral ventricle, and involving the corpus callosum secondarily. He gives as the characteristic symptoms of such tumours (1) headache and somewhat vague symptoms of progressive cerebral disease; (2) gradual onset of more or less well-marked hemiplegia; (3) appearance of similar symptoms on the opposite side of the body; (4) coming on of dementia with loss of speech, difficulty in swallowing, and want of control over rectum

and bladder. In this case all those symptoms were present, the only point in which a difference could be found being that, although the other symptoms were of gradual onset, both the initial right hemiplegia and also the later left one occurred suddenly.

The presence or absence of optic neuritis might be expected to help in the diagnosis of such a case, but unfortunately it is not so. Of the five cases related by Dr. Bristowe of tumour in or involving the corpus callosum, in two neuritis was present, in one it is noted as being absent at least ten days before death, while in the others it is not referred to. So that the absence of this condition in the present case (and there was no trace of it half-an-hour before death), did not give us much help. Nor could the absence of epileptiform convulsions count for anything, as in only one of Dr. Bristowe's cases were fits present.

The only alternative to such a tumour seemed to be some lesions in the pons probably of the nature of softening from occlusion of vessels. The suddenness of onset of the two attacks, the fact that after the second the right side of the face and the left side of the body were the parts most affected, and the absence of the peculiar mental change described by Dr. Bristowe in cases of tumour of the corpus callosum, were all in favour of lesion of the pons and against the probability of such a tumour. The idea of hæmorrhage into the pons at more than one point had occurred to us only to be discarded. As a rule one hæmorrhage into the pons kills almost at once, and it seemed most unlikely that two or more should take place into one pons giving rise to symptoms spread over two and-a-half years. Yet at the autopsy this was found to have taken place. There were in the pons hæmorrhages in such positions as to affect the tracts on the two sides, of which a more detailed description is given below. The corpus callosum was healthy, as also were the capsules and basal ganglia. The arterial walls were much thickened and the left ventricle was hypertrophied.

G. B., æt. fifty-three, married, policeman, admitted April 3rd, 1889; died April 24th, 1889.

Two years ago last January patient began to feel weak and to

lose flesh; continued to do so for a year. No definite symptom except occasional frontal headache. At the end of this time while at dinner one day he became sick, vomited, and would have fallen from his chair. He did not lose consciousness, and no twitching of limbs or face was observed. Immediately after, however, right arm and leg were found to be powerless, and face was drawn to right. Next day had severe headache, but had recovered so far as to be able to use knife with right hand, and two days later had so far recovered the use of his leg as to be able to walk downstairs. He has continued weak on right side since and has suffered a good deal from frontal headache.

Two months later, while walking with his wife, became "giddy" and caught hold of her. Had no loss of consciousness and did not vomit, but he was unable to speak for four hours. The right arm and leg seemed no weaker after this.

In December last had another attack similar to last. He was in bed, felt giddy; did not lose consciousness, was not sick; had no twitching, face was not drawn, did not lose any more power. Was unable to speak for half-an-hour.

His memory has been gradually failing during his illness, and during the last nine months he has become very stupid. Frequently takes no notice when spoken to, but has never been known to ramble. Has had almost constant headache, but only sickness once in addition to the occasion already referred to. This was in the out-patient room of the hospital seven or eight weeks ago.

During the last year has had frequency of micturition, and inability long to resist the call, as well as loss of control over liquid motions.

His previous health was excellent, and he was regular in his habits. He neither drank nor smoked, and his family history shows nothing noteworthy. He denies venereal disease.

On admission patient is seen to be big, strong and healthy in appearance. There is distinct, although by no means very great right-sided weakness, affecting arm and leg, but not the face. The tongue is protruded straight, and the palate is unaffected. Sensibility is normal except that touches on right upper arm and forearm are referred to a segment below, while those on back of hand and fingers are referred to a point slightly higher. Touches on left upper limb are referred to about half a segment below. Sense of position of right arm is defective, of left is accurate.

Legs are well-nourished but feeble, right being the weaker. Sensibility to touch, or sense of position are perfectly normal.

His gait is feeble and tottering, but not characteristically hemiplegic. Knee jerks are active and equal; ankle clonus is well marked on both sides, and the plantar reflex is much exaggerated and equally so on the two sides. The wrist jerks are equally well marked; the triceps is rather more marked on right side.

The pupils react normally, there is no ocular palsy, and the optic discs and fundi appear normal. Taste seems somewhat blunted, equally so on the two sides. Smell is acute.

There is evident hypertrophy of the left ventricle, and a slight systolic murmur accompanying first sound at the aortic cartilage where the second sound is sharp. The urine has a specific gravity of 1018, and contains a distinct trace of albumen.

As to his psychical condition, he is dull and slow in comprehending, his memory is bad, articulation is thick, and he has difficulty in finding words. He understands what he reads, and also what is said to him, but he takes some time to do so.

Patient remained in the same condition during the first week of his stay in hospital. On the morning of April 10th he was observed to be very weak on left side, and on examination the arm and leg on this side were found to be paralysed. He could only just move the hand, he could not raise it to his head, and his leg was completely paralysed. The tongue was protruded to the left, but the mobility of left side of face was distinctly greater than that on the right side. The temperature was normal. The speech was rather more indistinct than formerly. During this and several succeeding nights, and also sometimes during the day, it was observed that the respiration was of the Cheyne Stokes variety, but occurring during natural sleep. It began as gentle respiration, gradually increasing in force, then dying away again until there was entire cessation for about twenty to thirty seconds.

Patient gradually became more and more stupid, began to pass his evacuations in bed, and to have difficulty in swallowing. The right side also seemed to become more feeble. The optic discs remained distinct and the margins well defined.

On April 23rd the following note was made:—"Patient takes still less notice to-day. He opens his eyes when one goes to his bed side; when asked to put out his tongue takes no notice. Blinks when hand brought before either eye. Can be made to hold up right arm very slightly when it is passively raised from the bed. Does not take any notice when pricked on arms or legs, but does if pricked on his face. Plantar reflex brisk, left greater. Knee jerk not so active as formerly, left being greater; ankle clonus well marked on left side, slight on right. Temperature

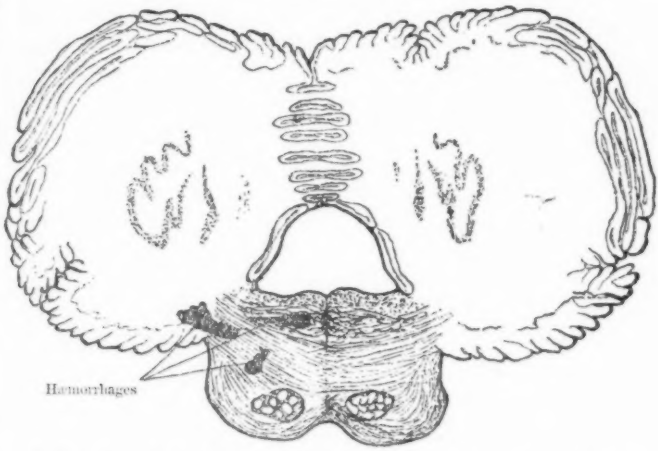
this morning is 102°. Urine acid, 1026, no deposit; marked cloud of albumen, no sugar. Passes evacuations in bed. Mr. Gunn examined eyes to-day and described discs as hyperæmic, the inner margin indistinct, but no effusion or other sign of inflammation."

Patient gradually sank into complete coma and died on the day after the foregoing note was made. Temperature rose to 104° before death.

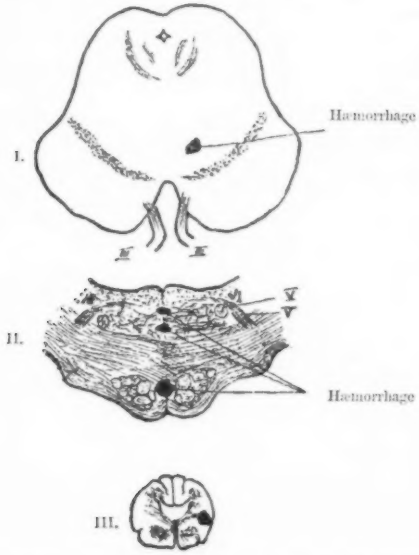
Post-mortem.—Heart was found to be large, left ventricle very much hypertrophied, valves all competent. Very slight atheroma of aorta. The vessels generally, but especially those in the brain, were very much thickened, and atheromatous patches were evident in several places. The kidneys to the naked eye appeared fatty. The capsule stripped off readily. The left had a urine-containing cyst about the size of a large marble just under the cortex. The other abdominal and thoracic organs appeared healthy.

In removing the brain the dura was found to be rather adherent to the skull. There was no flattening of the convolutions, and superficially there was no evidence of disease except the condition of the vessels already referred to. On section of the brain no softening or hæmorrhage was found in the hemispheres, nor could any blocking of vessels be discovered. The corpus callosum seemed normal.

The accompanying drawings which Dr. Colman has been kind enough to make for us show the condition of the parts about the pons. The figures represent the appearance of sections at different levels, between the crura and the spinal cord, and in all of them one or more hæmorrhages can be seen. It is worthy of note that several sections of the pons at intervening points show to the naked eye no sign of disease, the vertical extent of each hæmorrhage being very small.



Section through the pons and cerebellum shewing hæmorrhages into the right middle peduncle.



I.—Section through the crura cerebri at the level of the third nerve.
II.—Section through the pons at the level of the nucleus of the fifth nerve.
III.—Section through the medulla at the level of the decussation of the pyramids.

Clinical Cases.

A CASE OF BULBAR PARALYSIS WITHOUT STRUCTURAL CHANGES IN THE MEDULLA.

BY LAURISTON E. SHAW, M.D., M.R.C.P.

P. S., æt. thirty-seven, a baker, applied for treatment at the out-patient department at the Victoria Park Hospital, on Friday, February 11th, 1887. From statements he then made, and from facts obtained from his widow on a subsequent occasion, the history of the case is briefly as follows:

He was a married man with seven healthy children. He had always followed his present occupation, that of a baker; he had never suffered from any venereal complaint, and had enjoyed perfect health until his present illness began, six months ago. At that time he was kept from his work by a very severe attack of diarrhœa, which lasted a fortnight, but did not necessitate his staying in bed. Ever since this attack he had suffered from an ill-defined feeling of weakness, becoming gradually more severe, but with occasional remissions. This progressive weakness had been noticed more in the arms than in the legs, and had quite early in the illness been observed in the act of mastication. Throughout there had been wasting of the body generally, and at no time had the patient been well enough to resume his work. During the last month the weakness had been much more marked, and it was within this time that the symptoms had been noticed which led him to seek advice at the hospital.

There had been difficulty of articulation, so that the patient could not make himself clearly understood. At the same time he had found trouble in deglutition—a trouble that was more marked with solid than with liquid food. These symptoms were but slight in the morning, but became more evident as the day advanced. There had been copious secretion of frothy mucus, which he found very difficult to spit out, and which he feared sometimes would choke him. Within the last fortnight he had

two severe attacks of shortness of breath, which gave great alarm to his friends.

He walked into the out-patient room assisted by a friend and required help in undressing. He could however stand and walk a little unaided, but said that he often fell, and was quite unable to walk downstairs alone. When sitting he could not cross one leg over the other without lifting it with his hands. He spoke slowly and indistinctly, reminding one of a patient suffering from tonsillitis. The special difficulty of articulation was not investigated except that he had more difficulty in pronouncing hard "c" than other consonants. There was no nasal twang to his voice, and his soft palate was seen to move well and equally. His mouth was constantly held slightly open, but there was no dribbling of saliva, on the contrary what saliva and mucus collected in the mouth was cleared away with the fingers after futile attempts to expectorate it. There was loss of power in the lower facial muscles, so that the naso-labial fold was obliterated on either side, causing a vacant expression. He was unable to whistle or blow out a light. The loss of power was quite equal on the two sides, and did not affect the upper facial muscles at all, the patient being able to screw his eyes up firmly. The only evidence of any weakness of the muscles of the tongue was a difficulty in starting its protrusion; when protruded it could be moved in any direction, though slowly. The masseters were decidedly weak; the man stating that he was obliged to help mastication by pushing up his jaw with his hand, and complaining that he could not hold his pipe in his mouth either with his lips or his teeth without similar aid. The difficulty in swallowing of which he complained was well shown on his attempting to drink some water. The patient having filled his mouth, threw his head back, when some of the water seemed to get into his œsophagus, but almost at once he choked, and holding his head forward most of it ran from his lips. The laryngoscope showed his vocal cords to move well, whilst the ease with which the examination was made seemed to show some impairment of sensibility of the fauces.

Diplopia had sometimes been noticed by the patient, though at the time of his visit there was none; the pupils, motor oculi muscles and fundus of the eye were all normal.

The muscles of the trunk and extremities were ill-developed and flabby, but there was no obvious wasting of any group of muscles. The grasp was decidedly feeble, but equal on the two sides.

The wrist, elbow and knee jerks were brisk, a little above the average. In the right ankle an attempt to produce clonus resulted in four or five contractions; there was no response on the left side. The cremasteric and plantar reflexes were present and equal; the abdominal and epigastric absent.

His memory and other mental functions appeared normal. Throughout his illness there had been perfect control over his bladder and rectum.

Physical examination of his chest showed nothing abnormal with the heart or lungs. On first lying down for examination the patient complained of difficulty of breathing; no change in the rate of respiration or general condition could be observed, and the sensation soon passed off.

The man became an in-patient on Februray 12th, 1887. His condition did not attract special attention until the evening of the 13th. At that time he was attacked with sudden and urgent dyspnœa. The resident medical officer who was called to see him considered his condition to be due to paralysis of the intercostals, the expansion of his chest being carried on by the accessory muscles of respiration. He was becoming rapidly cyanosed, so that artificial respiration was tried with good result. The improvement was only temporary, and the proceeding had to be resorted to again and again. The following note was made of his condition in the intervals of the attacks: "His tongue is protruded straight. There are no ocular paralyses; pupils react readily and equally. He speaks clearly. The mouth is not drawn to one side. He cannot swallow his saliva, which collects about the fauces and is spat out with great difficulty, apparently from weakness of the muscles of the cheeks and tongue. Respiration is short and jerky; chest is full of large moist râles. Nothing can be felt at the back of the pharynx. There is no obstruction in the larynx to the entry of air. His hands are cold, and when an attack of dyspnœa is coming on he beats his chest and throws his limbs about in great distress, and asks to have artificial respiration performed. His mind is quite clear."

Subsequently the attacks became more frequent and severe, especially with regard to the urgency of the feeling which the patient described as an inability "to expand his lungs." Artificial respiration was repeatedly performed and for long periods, but this was at last ineffectual and the patient died a few hours after the beginning of the attack.

I made a post-mortem examination of the case on the afternoon of February 14th. Beyond some pus in the smaller

bronchial tubes and an enlarged mediastinal gland, every organ in the body was normal. Especial care was taken to search for signs of old syphilis: there was no scar on the penis and the testes were normal. The vertebral column was healthy, and nothing abnormal found in the upper respiratory passages.

To naked-eye examination the brain appeared perfectly healthy, as also did its meninges and those of the spinal cord. The pons, medulla and spinal cord, as being the likely seats of lesion, were handed to Dr. Newton Pitt for microscopical examination. The following is the report of the examination kindly sent to me by Dr. Pitt.

"Pons, medulla and spinal cord normal to naked-eye investigation. Specimen hardened in Muller's fluid, and sections stained some with aniline blue black, some with Weigert's hæmatoxylin stain. Sections have been made and examined from the following situations: through the fourth and first cervical segments, at the lowest part of the medulla, through the medulla just before the canal opens, *i.e.*, at the level of the eleventh and twelfth nuclei, and four others higher up of which one was through the vagal nucleus and one through the locus cœruleus. The cells of the nuclei, the fibres, vessels and all the tissues in every section appear normal, hence any organic disease of these parts of the nervous system may be safely excluded."

The clinical combination of defects of respiration, deglutition, articulation and mastication, naturally led us to expect the medulla to furnish us with a pathological explanation of the case. The failure to find such explanation adds one more to the many recorded and unrecorded cases in which physiological knowledge and previous pathological experience do not lead to a correct solution of the mystery of disease. Whether this is to be found by examining more carefully the suspected organ or tissue, or by looking elsewhere, or by seeking for possible causes of change in the function of the organ other than visible alterations in its structure can account for, is only to be determined by as far as possible adopting all these methods in every case. The advantage of reporting such negative cases as the one above seems to be that we are kept on our guard against assuming slight structural changes which may possibly be only secondary to the main disease to be its real cause.

A CASE OF POLIOMYELITIS CHRONICA OCCURRING IN A CHILD AGED FIVE YEARS.

BY F. W. KIRKHAM.

Mary H., a child, aged five years, was brought to me on the 4th September, 1886.

The patient had always enjoyed good health, and had had no previous illness of any description.

She commenced to walk when fourteen months old, her teeth appeared quite regularly. She had never suffered from convulsions.

For about a month before I first saw her she had been noticed to walk somewhat unsteadily, and her legs seemed after a little exertion to be weak and powerless. These symptoms continuing to increase; her parents consulted me about her. When I first saw the patient her appearance and general condition was that of a healthy child.

She was slight but well nourished, and seemed bright and intelligent. On being put upon her feet it was found she could stand with great difficulty, and on walking took hold of surrounding objects to steady herself. There was at that time no apparent wasting of the muscles of the legs or thighs, and the patellar reflex was normal on both sides.

The weakness was stated to be more marked in the left than in the right leg, but, on seating her on her mother's knee, there appeared but little difference between the condition of the two, the movements of both being extremely weak and uncertain.

There was no anæsthesia or paræsthesia, and the powers of micturition and defæcation were unaffected.

On applying the faradic current to the nerve trunks of the lower limbs there was very little response, and the same was noticed of the application of the same current to the muscles. A constant current up to forty Stöhrer's cells applied to the nerve trunks of the lower extremities produced but slight contractions,

a current of twenty cells applied to the muscles, however, produced slow and tonic contractions A C C=K C C.

I saw the patient occasionally during the next few weeks, but had not an opportunity to test further the electrical reactions till May 14th, 1887. At this time her condition was much worse than when I first saw her, she now being wholly unable to stand or walk, though she could move her legs slightly when sitting. There was also marked atrophy of the muscles of the calves and thighs.

There was no response whatever to very strong faradic currents, whether applied to the muscles or nerves.

There was also no response to the application of a strong galvanic current to the nerve trunks, but vigorous, slow and tonic contractions when a very weak current was applied to the muscles, A C C > K C C. The patient remained in this condition for about two months, when she was again seen by me, the parents then thinking they observed a slight increase of motor power in the paralysed limbs.

This appeared certainly to be the case, and when the child was again brought to me in November, 1887 there was a very decided improvement, she being able to stand when supported a little. There was now a little reaction when the faradic current was applied to the nerve trunks and muscles of the limbs, and of galvanism to the nerve trunks, but application of a weak constant current to the muscles still produced slow tonic contraction, A C C > K C C.

I did not see the patient again until the following February, when she had recovered the complete use of her legs, and seemed to be in perfect health.

The electrical reactions were then perfectly normal, and the wasted muscles had increased in volume and were nearly, if not quite, as well developed as in other children of her age.

She has since remained quite well.

A CASE OF HEMIATROPHY OF THE TONGUE WITH ITS PATHOLOGY.

BY E. F. TREVELYAN, M.D.(LOND.) B.SC.
Resident Medical Officer and Pathologist, Leeds General Infirmary.

ANNIE B——, aged twenty-five, was admitted into the Leeds General Infirmary on May 1st, 1889, under the care of Dr. Churton, to whose kindness I am indebted for permission to publish the case. The following clinical notes were taken by Mr. Walker, formerly house physician, and Mr. McCandlish, the clerk of the case :—

The patient, a stout well-built woman, weighing nine stone, complains of pain in the neck, wasting of half the tongue, and chronic abscesses below the jaw. Temperature 99° ; pulse 120 ; respiration 26. The pain in the neck is increased by even slight pressure on the cervical spine ; the pain is very severe if the vertex of the skull is pressed upon. When asked to turn her head she turns her body instead, keeping her head fixed. She has some pain in the left ear on swallowing ; her hearing is good. She also complains of pain over the left eye. No affection of first, second, third, fourth and sixth cranial nerves. The left half of the tongue is markedly atrophied ; it can be moved from side to side both in the mouth and when protruded, but not so easily in the latter case. The left half of the tongue is felt to be thin from wasting of the lingualis. The organ is exactly represented by the drawing in Gower's "Diseases of the Brain" (vol. ii., p. 278). There are marks of former strumous glands on the left side of the neck, two burrowing fistulous sores below the jaw on the right side. The thoracic and abdominal viscera are healthy.

She gave the following history. Seven years ago she had suppurating glands in the neck, but was otherwise quite well up to eight weeks ago. At that time she went out one night and on her return had pain and stiffness in the left side of the neck. This was looked upon as a simple neuralgia, but it did not yield at all to treatment. Three weeks ago she was sent to Har-

rogate, got a chill, and next morning her tongue was swollen, and shortly after this she noticed the left side of the tongue began to waste. Mr. W. H. Brown was called in and recommended her removal into the infirmary. A few days after admission Dr. Churton tested her taste with dry quinine, salt and sugar. There appeared to be *no* taste on the left half of the tongue, either at the top or in the middle, nor even on the palate. The pain in the neck, with inability to move the head or the neck in any direction, continued. She could not wear a support for the head on account of the suppurating glands in the neck. The temperature both morning and evening was high. On June 18th the pain was worse and she began to vomit and to be rather delirious, calling for her father and taking anyone who came to her bedside to be her father, otherwise she was inattentive to what was going on around her. On the same evening she had a fit, foamed at the mouth and bit her tongue. Her face and arms twitched during the fit and she had marked Cheyne-Stokes breathing after it. The face and arms twitched occasionally during the night. Next morning (19th) another fit occurred, the right arm and both sides of the face being convulsed. The pupils were dilated, the face dusky. Temp. 102°. She was quite unconscious. She remained so during the day and died at ten p.m.

The following is an account of the morbid anatomy. The left side of the tongue is much atrophied.

Occipital Bone.—In the neighbourhood of both occipito-atloid joints the bone is much roughened and worm eaten. No definite collection of pus was found there.

Atlas.—Superior articular surfaces are both diseased, the left being the worse of the two. The cartilage covering them is worn away in parts and distinctly honeycombed in others. This is more marked on the left side. These articular surfaces are less distinctly defined than they should be, a distinct rough surface of bone running round the left one. The bone is also seen to be rough and uneven on the anterior surface of the anterior arch on the left side. There has been clearly a deposit of new bone here. The rest of the bone is healthy. The transverse ligament was found entire.

Axis quite healthy except the upper part of the odontoid process, which is roughened. A section taken through odontoid process shows healthy bone. The rest of the cervical spine is healthy.

Base of Brain.—There is considerable thickening of the men-

inges at the base of the brain (especially of the pia mater and arachnoid). This is very noticeable about the origin of the left hypoglossal nerve, its two bundles of fibres passing through the thickened membranes. No such thickening can be made out round about the right hypoglossal nerve. There is no tumour of any sort at the base of the brain (nor was there any in the upper part of the neck in the course of the hypoglossal nerve. Patient had suffered from chronic inflammation in the glands of the neck). Sections taken at varying levels through the Medulla Oblongata show no change in the hypoglossal centres. The cells are well developed and no difference is noted between the two sides. No change is visible in the infra-nuclear tract.

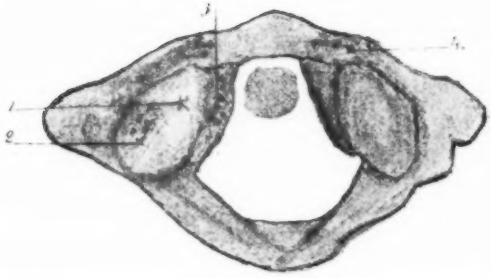
Nerves.—Left hypoglossal is undoubtedly smaller and more shrivelled than right. A section treated with osmic acid and stained with *Picro-carmine* presents the following appearances under the microscope:—The nerve is very irregular in outline, presenting projections and depressions all round its circumference. The epineurium is greatly thickened and bands of connective tissue are seen making their way inwards from the circumference. When the section is examined by a low power fasciculi of nerve fibres are seen scattered irregularly throughout the section. In one place they are more abundant, in another hardly any are to be seen. In between these fasciculi the connective tissue has undergone very considerable increase. It forms a branching net-work enclosing the fasciculi, or what is left of them. It is seen to contain a number of fusiform fibres. Under the high power it may be seen that many fasciculi have perished, leaving hardly any trace of their presence, whilst others are in the process of disappearing. In between the fibres themselves the endoneurium is increased and individual fibres are seen gradually undergoing atrophy.

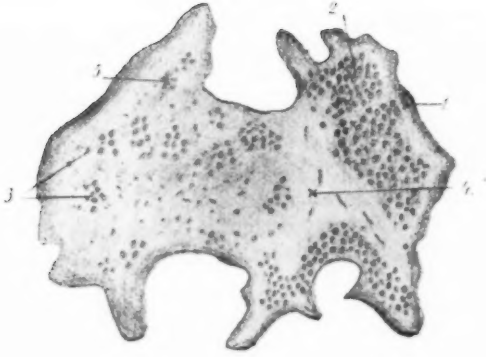
Right hypoglossal nerve is healthy.

Right and left gustatory nerves are healthy.

Left chorda tympani is quite healthy. This nerve was examined at the instance of Dr. Churton, with relation to the loss of taste on the left side of the tongue.

Hemiatrophy of the tongue is a rare disease, so that it seldom occurs that a case can be reported in its entirety, viz., with an account of the *post-mortem* examination appended. The chief interest of this case lies in the fact that the hemiatrophy was due to one of the rarer causes of this disease (viz., involvement of the nerve in thickened meninges), and that this cause





was made out and verified by *post-mortem* examination. Dr. Jacob, the Honorary Pathologist, examined the body after death, found that basal meningitis was the fatal lesion and that the occipito-atloid articulation with the neighbouring occipital bone was diseased. A more exact description of the affected parts has been given. The history of the case was the usual one of disease of the upper cervical spine—a history of deep-seated pain in the region of the neck, at first supposed to be a simple neuralgia—followed by rigidity of the neck. Five weeks from the beginning of the disease the left half of the tongue began to waste.

On admission into the infirmary she had all the ordinary signs of cervical caries. No other cranial nerves were involved, except in so far as taste was concerned. There was no paralysis of the palate. (Unfortunately a laryngeal examination was not made, but the absence of signs, the site of the disease and the non-involvement of the spinal accessory nerve make it practically certain there was no laryngeal palsy.)

It is proposed here to enumerate the causes of hemiatrophy of the tongue, and to illustrate them from cases which have already been published. We have then to consider the pathological changes which may affect: (1.) The nucleus of origin of the hypoglossal nerve or its fibres as they pass out through the M.O. to emerge between the pyramidal and olivary bodies. (2.) The nerve from where it leaves the medulla in two bundles until it quits the anterior Condyloid foramen as a single trunk, and thirdly and lastly, the trunk of the nerve from the anterior condyloid foramen to its final distribution in the tongue.

(Disease of the hypoglossal path. from its nucleus of origin in the M.O., up to its representative in the cerebral cortex, can hardly give rise to a wasting out of proportion to disease, viz., to such wasting as we are concerned with here.)

(1.) The nucleus of origin and infra-nuclear tract in the M.O. may be affected by (*a*) hæmorrhage, (*b*) softening, or (*c*) degeneration.

(*a*.) A remarkable case which illustrates atrophy of the tongue as a result of hæmorrhage into the M.O. is related by Dr. Hughlings Jackson (*Lancet*, 1872, p. 771). The atrophy was here curiously bilateral.

The patient, fifty-two years of age, was suddenly seized

while recovering from hemiplegia, with paralysis of the tongue. The tongue subsequently atrophied. There was no laryngeal palsy. *Post-mortem.*—Dr. Lockhart Clarke found among other lesions the remains of a hæmorrhage into the left olivary body.

(*b.*) The second process that may affect the nerve and its centre in the M.O., is softening. Thus Prof. Hirt (*Berl. Kl. Wochenschr.*, 1883) tells of a patient who was seized with an apopletic attack. One month later the right half of the tongue began to atrophy. The right recurrent nerve was paralysed. Condition of palate is not mentioned. Taste normal. The lesion was put down as bulbar hæmorrhage with embolic softening of the centres of the hypoglossal and spinal accessory nerves.

(*c.*) The third morbid change is degeneration. Raymond and Artaud relate (*Arch. de Physiologie*, vol. xvi., pp. 367) a most interesting case. A patient suffering from tabes dorsalis for eight years, began to have difficulty with his speech one year before he came under observation. At this time he had well marked right hemiatrophy of his tongue. Eight years later he died. The cells in the principal nucleus of origin of the right hypoglossal nerve were found degenerate and pigmented.

As regards hemiatrophy of the tongue in tabes dorsalis, M. Ballet has pointed out (*Arch. de Neurologie* vii., pp. 191, 1886) that it may come on early in the disease, that it may be associated or not with ocular palsies or atrophy of some of the muscles of the extremities. This was so in MM. Raymond and Artaud's case, where there was slight wasting of the thenar eminence. More recently Plücker in an inaugural address at Würzburg (*Centralblatt f. Laryngologie u. Rhinologie*, Oct., 1889) reported a case of tabes where left hemiatrophy of the tongue existed early in the disease. Atrophy of half the lower jaw was also present in this case.

This lesion, viz., degeneration may not be limited to the hypoglossal centre, but may involve the neighbouring nucleus of the spinal accessory nerve (the palate being paralysed at the same time), thus giving rise to what has been called associated paralysis. This has nearly always occurred in

syphilitic patients. The exact lesion has apparently hardly ever been proved by *post-mortem* examination, and therefore it cannot be said for certain whether it is a degeneration such as occurs in other parts of the nervous system in syphilitic subjects or whether on the other hand, it is a softening—the result of thrombosis, the latter again being caused by a syphilitic endarteritis. Thus Dr. P. K. Pel relates (*Berl. Kl. Wochenschhr.*, 1887) a case of a man, æt. 34, who had left hemiatrophy of the tongue, paralysis of soft palate (left) and left vo. cord. Patient had had syphilis. He had knee jerks, but there were no other tabetic symptoms. Dr. Pel attributes (without *post-mortem* evidence) the disease to softening of the hypoglossal and left vago-accessorius nuclei due to thrombosis of the left anterior spinal artery, a refinement of diagnosis to say the least of it. Several cases of this associated paralysis (half the tongue and palate and the vo. cord of the same side) have been reported from time to time, by Dr. Hughlings Jackson, in the *Brit. Med. Journal*, 1887 (Right). *Lancet*, 1886 (Right), *Lond. Hosp. Reports*, by Dr. S. Mackenzie, *Brit. Med. Journal*, 1883 (left), and *Clin. Soc. Trans.*, 1886. In the second of Dr. Mackenzie's patients there was no history of syphilis, the man attributing his illness to the fumes of Hg., Pb. and Sb., among which he had to work. In some of these cases some other muscles have been paralysed, as for instance, the Trapezius.

Leudet (*Ann. des Mal. de l'Oreille, Dec.* 1887) has also related two cases of associated paralysis with histories of syphilis, none of tabes. Mr. Holthouse reported a case of paralysis of the sixth and ninth nerves in a syphilitic patient. The atrophy (right sided) was hardly apparent in six years time. This case is put in here on account of the affection of the two nerves in a syphilitic subject. The progress of the case and the nerves involved would make it probable that the disease was not of central origin.

Some few cases have been reported of progressive facial hemiatrophy, in which there has been atrophy of the tongue on the same side, for instance Dr. Buzzard's case (*Clin. Soc. Trans.* V.) with right lingual hemiatrophy. Dr. Dreschfeld's case (*Brit. Med. Jour.*, i., 503, 1882) with left hemiatrophy.

Dr. Payne's case (*Brit. Med. Jour.*, 1880, i., p. 743) with left hemiatrophy. On the other hand some cases of this progressive hemiatrophy have been recorded where there has been no atrophy of the tongue. Thus, Prof. Berger (*Deut. Arch., f. Klin. Med. Band.*, xxii., p. 432) tells of a boy who had right hemiatrophy of the face after a fall, in which there was no atrophy of the tongue.

(2.) The hypoglossal nerve may be involved in disease between its exit from the M.O., and where it leaves the anterior condyloid foramen.

(*a.*) It may be involved in thickened meninges as in the case of Hayem and Girandeau (*Révue de Medicine*, 1883). Dr. Churton's case is also a good example of this, the nerve being involved in a thickening of the Meninges which itself was consequent upon disease of the occipito-atloid joint and neighbouring bone.

(*b.*) The nerve may be pressed upon or embedded in a tumour. In the classical case of Dupuytren, a hydatid cyst had inserted itself into the anterior condyloid foramen. The late Dr. Habershon (*Med. Times*, Feb. 9th, 1881) relates a case where a secondary growth involved the basilar process and extended into the right temporal fossa, involving the eighth and ninth nerves. The sterno-cleido-mastoid muscle was also affected.

A case is reported by ROMBERG (*Nev. D. Syd. Soc. Trans.*, p. 307) where there was probably pressure on both hypoglossal nerves at their roots of insertion into the M.O., this giving rise to complete atrophy of the tongue.

Mr. FAIRLIE CLARKE reported a case of hemiatrophy of the tongue, which was caused by a secondary tumour within the brain or high up in the neck (*Med. Chi. Trans.*, vol. vii., No. 1, 1872.)

A most interesting case has been reported by LEWIN (*Jahres. Gesamt. Med.*, 1883) where the hypoglossal nerve (left) was involved in a gumma which was seated in front of and in the anterior condylar foramen. The patient had left lingual hemiatrophy. There was diminished tactile sensation and sensation to heat and pain. Taste perceptions lessened. There was salivation. There were also gum-

mata at the root of the tongue. Nucleus of hypoglossal nerve healthy.

(*c.*) The nerve may be involved as it passes through the anterior condyloid foramen by thickening or cancerous disease of the bone.

A very interesting case is related by Dr. HUGHES BENNETT, where the whole tongue was atrophied, but the case is so important that it can hardly be passed over in any account of hemiatrophy of the tongue. Patient, æt. 54, had a tumour removed from the breast; eight years later she had atrophy of the tongue. She had peripheral disease of the facial nerves as well as of the hypoglossal. At the *post-mortem* examination numerous patches of degenerated bone due to malignant disease were found within the cranium, varying from a pea to a shilling in size. On careful examination the internal auditory meatus and anterior condyloid foramen were involved on each side, in such a way as to cause pressure on the nerves and peripheral degeneration. The symptoms resembled bulbar palsy in life, the reaction of degeneration being one of the chief distinguishing points.

(3.) The nerve may be damaged in its course from the anterior condylar foramen to its final distribution in the tongue, by (*a*) injury, or (*b*) disease.

(*a.*) Dr. HYDE SALTER (*Encyclop. of Anatomy and Phys. Art. Tongue*), relates the case of a man who was stabbed in the neck. Both hypoglossal nerve and carotid artery were divided. Left hemiatrophy followed. Bernhardt (*Deut. Archiv. f. Klin. Med. Band.* xxii., S. 392), that of an attempted suicide who divided his hypoglossal nerve.

WEIR MITCHELL (*Injuries to Nerves*, p. 335), that of a lad who had a gunshot wound which injured the hypoglossal nerve, hemiatrophy of the tongue followed.

Much more recently Mr. RUTHERFORD MORRISON (*Brit. Med. Jour.*, 1888), has put on record the case of a girl who strained her neck in jumping over a grave. Paralysis (followed by atrophy) of the right half of the tongue ensued. Morrison attributed the disease to a hæmorrhage into the sheath of the hypoglossal nerve or else to a stretching of the nerve over the long process of the atlas.

Sir JAMES PAGET, long ago in the *Clin. Soc. Transactions*,

related a most interesting case of a young man who fell heavily upon his head. Paralysis of the right side of the tongue with wasting followed. An abscess formed and discharged. Dead bone was removed and the man recovered.

ERB, in his article on paralysis of the hypoglossal nerve (in the *Arch. f. Klin. Med.* xxxvii., p. 270) mentions that cases of paralysis with wasting have occurred as a result of dislocation of the atlas. These have been related by Uhde Hagemann and Böttche in Langenbeck's *Arch. f. Chirurgie.*

(*b.*) Hemiatrophy of the tongue may be caused by the nerve being involved outside the skull in growths, or even in enlarged glands, as related by Mr. Hutchinson (*Med. Times*, 1880, vol. 1. p. 57) in a case of malignant growth, and by Prof. Bernhardt (*Arch. f. Klin. Med. B.* xxii., S. 392) in a case of glandular enlargement.

Again, there seems to be no reason why the hypoglossal nerve should not be the seat of a neuritis, and this in turn, be followed by lingual hemiatrophy. Erb in the place cited above, relates a case of the disease of the hypoglossal nerve. After a full and admirable discussion he comes to the conclusion that the case was one of peripheral neuritis of rheumatic origin.

It may be well to conclude this paper by relating a case of great interest recorded by REMAK (*Centralblatt f. d. Med. Wissensch.*, 1886) in which there was a right hemiatrophy of the tongue in a case of lead poisoning.

Patient, æt. 43, a worker in lead, and addicted to alcohol had right wrist-drop and right hemiatrophy of the tongue. Pupils did not act to light. There was some limitation of ocular movements to the right. Paralysis of right post crico aryt. M. and Thyro arytenoid M. Knee jerks present, no signs of tabes.

The presence of Pb. poisoning is certainly suggestive that the lesion might be of peripheral origin, but again, the fact of its being associated with paralysis of the right cord receives an easier explanation if it be supposed to be of central origin. The close approximation of the centres of the hypoglossal and spinal accessory nerves easily explains the fact that a morbid process might spread from one nucleus to the other, and this appears to be a less strained explanation than one which supposed these two nerves (not at all susceptible to inflammatory change) to be independently the seat of a neuritis.

REMAK states that he believes the disease to be of central origin, and the excuse for introducing the case here must be that the behaviour of cases of lead poisoning is usually such as to suggest a peripheral rather than a central lesion.

CASE OF GENERAL PARALYSIS COMPLICATED BY APHASIA.

BY C. PRICE TANNER.

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I AM indebted to Dr. E. Marriott Cooke for permission to publish the notes of the following case :—

T. R., æt. thirty-three, married, telegraph clerk, was admitted into the Worcester County and City Asylum September 19th, 1889. The history obtained from his wife was to the effect that he had always enjoyed good health, and had had no illness till twelve months before admission. At that time he suffered from "writer's cramp," was low spirited, and seemed generally ill and out of sorts. He went for change of air to Lancashire, and after two or three months had apparently quite recovered. He remained well till August 24th. On that day, about 9·30 a.m., he was about to read the paper aloud to his wife, when he suddenly began to stammer and "talk jargon." He was put to bed, and soon afterwards his right arm and leg were found to be paralysed, and his mouth seemed to be drawn to one side. He was unconscious, and remained in this state till two a.m., August 28th, when he began to try to talk. His language, however, was quite incomprehensible. From that date till the time of his admission the paralysis has gradually improved, the improvement starting in the leg before the arm, but his speech has never become more intelligible, though he can utter more words now than he could at first. After being in bed about a fortnight he got up, and then was noticed an entire change in his moral character. From being a fond father he seemed to take an aversion to his children, and was found by his wife more than once ill-using them. On one occasion he was holding one of them on the floor, kneeling over her with a knife in his hand. He also became very suspicious, and since his attack has always slept with a hammer and knife by his bedside. He has also lately had extra locks and bolts put on his doors. In consequence of his threatening behaviour to his wife

and children it was found necessary to put him under restraint. He has had five children, all of whom are alive and healthy. There is nothing in the family history bearing upon the case in any way.

In the certificate upon which he was admitted into the asylum, it was stated that he was constantly muttering to himself, wandered about in an aimless manner, had threatened his wife with a poker, imagined he was telegraphing up the chimney and listened for a reply. He was stated to be epileptic on the strength of the "fit" of August 24th. *On admission*, he was found to be thin and poorly nourished, but free from bruises or marks of injury. All his thoracic and abdominal organs seemed to be acting healthily. There was slight tremor of the facial muscles; pupils equal; tongue deviated a little to the left side. The right limbs were weaker than the left, showing signs of the late hemiplegia. No wasting of muscles. Speech very aphasic (*vide infra*). Mentally he appeared to be in a condition of mania. Was extremely restless and agitated, wandering up and down the ward in an aimless manner, chattering to himself, and constantly trying to undress himself.

September 25th.—His aphasic symptoms are as follows:—He is quite unable to understand spoken words, except when asked his name. Cannot read printing aloud, though he can tell when the book is held the wrong way up. Cannot repeat spoken words, write at dictation, or copy printed matter. Can understand written words to a fair extent, and read them aloud; but in doing so makes mistakes in the pronunciation of letters, *e.g.*, calls "name," "tame." In copying writing frequently transposes the letters, but puts them all in, copying in this way even words spelt wrong, *e.g.*, he copied pensil as "pensli." He recognizes objects by sight, but apparently not by hearing or smell (touch?). He can articulate, but uses wrong letters, calling knife, "life," &c. The time 4·40 he calls "forty—forty-one—one." Can write his name correctly, but cannot pronounce it. On being asked (in writing) his age, he wrote down "about nineteen years of age." After careful testing no hemianopsia could be discovered.

October 10th.—At times seems very much frightened when spoken to, and on one occasion recently began to cry when given a pencil. His aphasia has slightly improved, and he can utter a few sentences and write a few words. His tongue has been noticed to be very tremulous when protruded. Has been put on a mixture containing hyd. perchlor. and pot. iod. tentatively.

November 3rd.—His aphasic symptoms vary a good deal from

day to day; sometimes he will write a few words, but rarely. At times appears very much frightened and distressed. He knows his bed and his place at the table, &c., and makes himself useful about the ward to a limited extent. Is very clean in his habits.

November 23rd.—He appears to be more demented. He does not realise where he is, and seems lost generally. He will sit still in one place for hours at a time doing nothing, and making no effort to amuse or occupy himself. Constantly makes a humming noise to himself, and if made to desist starts again almost immediately afterwards. When his wife or children are mentioned to him (in writing) he does not seem to take the slightest interest in them.

December 2nd.—On the 28th ult. he had a succession of thirteen fits in about five hours. He was apparently dying, and was with difficulty revived by enemata of brandy and chloral hydrate. The fits began in either one or the other hand, and then spread to the whole body; the head and eyes rolled slowly from side to side, generally to the left first. He is now confined to bed; both legs are completely paralysed and he is passing all his dejecta into the bed. A bedsore is beginning to form over the right trochanter. The knee-jerks, which were exaggerated after the fits, are now absent. Slight clonus of right ankle.

December 4th.—Both legs are becoming contracted at the hips and knees. Knee-jerks and ankle clonus entirely absent.

December 7th.—In spite of everything which could be done, the bedsore has spread in a most malignant way, and to-day a large slough has separated, leaving a deep unhealthy ulcer, almost exposing the femur.

December 10th.—Sores forming over left hip and sacrum; tongue dry, coated. Patient tries to pull off the dressings.

December 11th.—Sores beginning to form over both heels.

December 17th.—To-day began to refuse food. Is coaxed to take beef tea, milk and brandy. Restless, pulling off his dressings unless closely watched.

December 31st.—Since last note has grown gradually weaker; the sores have increased in area and depth. For the last two or three days has only been kept alive by the frequent administration of brandy and milk in small quantities. He died this evening.

Permission could only be obtained for an examination of the brain.

Head.—Skull-cap regular, of medium thickness and density.

Dura mater not unduly adherent to bone. No surface hæmorrhages. Arachnoid and pia mater thickened, opaque and milky. Large quantity of fluid in meshes of membranes. Adhesion and decortication well marked over both convexities, principally over the ascending motor convolutions of the two sides and the frontal convolutions of the left side. Brain generally wasted; convolutions flattened externally. White matter pale and firm to the knife. Grey cortical matter thin and anæmic. Ventricles enormously dilated, full of clear, pale, serous fluid. The amount of fluid that escaped after removal of the brain was at least seven ounces. Frosting present in lateral ventricles. Foramen of Monro dilated to the size of a threepenny piece. Centres of right side appeared healthy; those of left side compressed laterally. The whole of the left insula was destroyed, apparently the result of a hæmorrhage, its place being occupied by a mass of broken-down blood-clot, apparently becoming encysted. The hæmorrhage had invaded the first temporo-sphenoidal lobe near its tip, and also the part of the inferior parietal lobe, where it is adjacent to the fissure of Sylvius. This was the only point at which the lesion appeared on the cortex of the hemisphere. The surrounding parts were stained with blood pigment. Extending internally, the lesion had destroyed the claustrum, whilst the external capsule, lenticular nucleus and internal capsule were flattened in the direction of the lateral ventricle. The source of the hæmorrhage could not be traced. No other gross lesion existed. The cerebellum, pons and medulla oblongata were normal to the naked eye. No frosting in the fourth ventricle. Slight atheroma of the arteries at the base of the brain.

Weight of Brain.—Immediately after removal, 48½ ounces; stripped of its membranes and free from fluid, 36¾ ounces.

Remarks.—The above case presents, I think, features of interest, both on account of the obscurity of some of the symptoms, and the difficulties attending a correct diagnosis. The history clearly pointed to a cerebral lesion in the left hemisphere, probably cortical or sub-cortical in situation. Of the cause of this lesion, beyond the fact that it was almost to a certainty vascular, no evidence could be obtained. The fact of the paralysis clearing up in the limbs whilst the aphasia remained would limit the situation of the lesion to the neighbourhood of the convolution of Broca, that centre having been permanently injured or destroyed, whilst the centres for the limbs had been merely inhibited. The

aphasia was both motor and sensory—the latter including almost total word-deafness and word-blindness to a slighter extent. The co-existence of word-deafness with word-blindness would indicate lesion of the temporo-parietal region, as explained in a paper by Dr. Allan Starr, of New York, in a recent number of BRAIN;[1] and *post-mortem* appearances justify that conclusion in the present case, as the first temporo-sphenoidal convolution and—to a less extent—the inferior parietal lobule were involved in the lesion. The absence of hemianopsia would exclude the angular gyrus from participating in the lesion, though its association tracts must have been damaged, as was proved by his inability to read aloud. The loss of power to recall the names of recognised objects indicates, in a similar manner, lesion of the association tract between the occipital and temporal areas, and as the inferior parietal lobule was involved, this case also agrees with Dr. Starr's statement.[2]

Another point of interest is the extreme rapidity with which the symptoms of general paralysis, when once established, ran their fatal course. From the date of admission to the patient's death was barely fifteen weeks, and for the first three or four weeks of that time, at least, the symptoms were at best equivocal. Though his case was looked on with suspicion from the time of admission as possibly one of general paralysis, it was impossible to point to any group of symptoms that would indicate that condition with certainty.

Apart from the physical symptoms, however, this case also presented unusual difficulties with regard to the mental state of the patient shortly after admission, which led to some amount of correspondence with the Commissioners in Lunacy. Judging from the previous history, the man was undoubtedly a proper person to be put under care and control and detained in an asylum, yet for some weeks after his admission (after he had once settled down in his new quarters) his insanity was by no means obvious, and not until the bodily symptoms left no doubt as to the grave

[1] BRAIN July, 1889, p. 95. [2] BRAIN. July, 1889, p. 92.

nature of his malady, did his mental state—as exhibited in the asylum, and judged apart from the previous history—warrant a diagnosis of insanity.

I can only regret that a more complete examination *post-mortem* could not be made. The fulminating character of the trophic changes might have been explained by an inspection of the spinal cord, and an examination of the other viscera might have cleared up the mystery surrounding the origin of the cerebral lesion.

Critical Digests.

CHRONIC HYDROCEPHALUS.*

BY M. ARMAND RUFFER, M.A., M.D.

Most historical sketches of diseases begin by a reference to the father of medicine, Hippocrates, and by a quotation of his views on the subject to be discussed. Strangely enough this author, who has given graphic sketches of many strange and rare diseases, does not mention hydrocephalus in any of his works. After a search in the original, with the help of Littré's[1] translation, no reference to it could be found. It is true that, writing of epilepsy in the sixth volume of his works, he says[2]—"For when the disease has lasted long it is curable no longer, the brain being eaten away by the fluid and melts; the part thus melted is converted into water, which surrounds the brain and bathes it, causing the attacks to become more frequent and more easy," but it is doubtful whether this refers to chronic hydrocephalus or not. The lines in vol. vii. p. 27,[3] in which he talks of water forming on the brain, evidently refer to meningitis. It is impossible therefore to say for certain whether Hippocrates knew anything about chronic hydrocephalus.

Galen,[4] (Book xiv., page 782) states that:—"There are four kinds of hydrocephalus: the first between the brain and meninges; the second between the meninges and the bone; the third between the bone and the pericranium; the fourth between the bone and the skin. The hydrocephalus between the skin and the pericranium we treat with two or three free incisions; that between the meninges and the brain is incurable." It is not possible to say whether "*ἰὰ μέν μειυξὺ*

* Abstract of Thesis written to obtain the Degree of Doctor of Medicine of the University of Oxford, June. 1888.

ἐγκέφαλου κὰς μήνιγγος" refers to water contained in the brain itself or present between the brain and the membranes covering it. In any case his knowledge of the subject was extremely vague.

Celsus[5] gives the following account of hydrocephalus:—"Praeter haec etiamnum invenitur genus quod potest longum esse ubi humor cutem inflat, eamque intumescit et prementi digito cedit: *ὑδροκέφαλον* Groeci appellant. "Besides these there is also found a kind which may be of long duration, and where the fluid swells up the skin, raises it and gives way under the pressure of the finger, the Greeks call it hydrocephalus."

Oribasius,[6] living in the 4th century, says that:—"The disease called hydrocephalus is the result of the compression of the head by midwives during the birth of the children. The patients afflicted with this disease present on the head a considerable tumour, elongated in form, giving rise to no little disfigurement. It is impossible to have an accumulation between this membrane (meninges) and the brain itself, for an individual suffering in this way would die before the disease called hydrocephalus had time to develop itself!" The first part of this quotation evidently refers to cephalo-hæmatomata, whilst the second sentence shows that Oribasius was ignorant of the existence of the disease now called hydrocephalus. Further on, he adds that "When the liquid is accumulated between the membrane of the brain and the bone, there will undoubtedly be a tumour, for in young children the bone not yet being ossified moves about, is easily depressed, and hence is easily distended by the pressure of the fluid; but one may see, at least, a solid painful tumour, which elongates into a sharp point. One finds the regions of the forehead and temples thinner than would be expected, having regard to the development of the body, for the parts in this region become contracted, being drawn upwards. These patients have flashes before the eyes and blinking of the eyelids!" The first part of this second quotation may possibly refer to meningo-encephaloceles, and the last shows that he was aware of the existence of nervous symptoms in these patients.

Hildanus de Hilden[7] relates three cases of the disease, and Vesalius[8] has given an account of one. Their accounts refer to young children, whose heads slowly and gradually filled with water, became of monstrous volume, the increase being due not only to widening of sutures, but also to the undue development and widening of the bones of the cranium. This last mode of growth was, in Vesalius's opinion, sufficient to account for the widening of the head in one case; the cranium acquired the circumference of two feet and ten inches in the course of fifteen years. In the three cases of Fabricius[9] the *post-mortem* examination showed that the head had been converted into a membranous cyst, which gave issue to a large quantity of water, amounting to eighteen pounds in one patient.

This subject could not escape the acute power of observation of Ambroise Paré.[10] In his works he first gives an account of hydrocephalus outside the cavity of the skull, attributing to traumatism a large share in its production, and after discussing Vesalius's case of true hydrocephalus, he goes on to say:—"Or, lors que cette humeur est entre le crâne et le dure-mère, ou aux ventricules du cerveau, et en toute la substance, le malade a les sens hébétés, comme l'ouye et la veue, et quand on presse sur la tumeur, elle ne s'enfonce, pas aisément . . . . Albucasis escrit avoir veu un enfant duquel la teste s'agrandissant tous les jours par l'aquosité contenue dedans, enfin devint si grosse, qu'il ne la pouvait soutenir debout, ny assis, et mourut quelque temps après. J'ai veu quatre enfants malades de cette affection de la teste, de l'un desquels je fis section après sa mort, et proteste n'avoir trouvé de cerveau non plus gros qu'un esteuf, et jamais n'en ai veu un seul guérir, lorsque l'aquosité est en grande abondance aux parties intérieures de la tête, mais lors qu'elles est aux parties extérieures on reçoit guérison. Donc si l'humeur est seulement entre le cuir musculeux et le péricrane ou entre le péricrane et crâne, si elle est petite faut essayer à la résoudre, et si l'on ne peut il faut faire incision, et fait sortir l'humeur, évitant le muscle temporal et faire sortir l'humeur, qu'on trouve comme une laveure de chaire sanglante: autrefois de sang noir: et si elle est causée de contusion, on trouve

avec le dit humeur du thrombus de sang. Ayant fait l'ouverture, et l'humeur étant évacué, on remplira la playe de charpie sec, et sera mis par dessus une compresse faisant ligature propre; et le reste de la cure se parfera comme il sera nécéssaire."

Morgagni[11] in his classical work, *De sedibus et Causis Morborum*," gives the result of several autopsies of cases of hydrocephalus as well as of some *post-mortem* examinations on congenital absence of the brain and spinal cord, encephaloceles and spina bifida. He, at the same time, mentions several authors who had written on the subject. This chapter is one of the most interesting in this classical work, for after most accurate descriptions of the pathological specimens, Morgagni bases on these facts theoretical considerations to explain their occurrence. Among the authors quoted by Morgagni are Hunauldius, Hildanus, Paisenius, Pechlinus, Stalpartius and Brinius.

The following are among the interesting cases mentioned by Morgagni. In the first, diminution of the hydrocephalic head followed on tapping a spina bifida, whilst, in the other, the cure of a spina bifida was followed by an increase in the size of the head. In another part the same author states that it does not follow that because a patient has a spina bifida, hydrops of the head must necessarily be present also. This statement shows that even in his time, the existence of a relation between the two diseases was an established fact. We may conclude therefore that in the 16th and 17th centuries, the existence of a disease corresponding to what is now called chronic hydrocephalus was undoubtedly established.

Yet neither Ambroise Paré, the greatest accoucheur of his time, nor, according to Poullet, any of the authors before the 18th century, mention hydrocephalus as a cause of complication in labour.

Wepfer[12] of Schaffhausen in 1665, saw two cases of fœtal hydrocephalus giving rise to dystocia, but unfortunately his paper on the subject was not published till 1727, long after his death. In the first edition of De la Motte's[13] work there is an account of two cases of undoubted hydrocephalus

causing dystocia, one being a head and the other a breech presentation. Smellie,[14] however, was the first to attract, in 1752, universal attention to the subject. He not only published several cases but also perforated the fœtal head in one case and laid down definite rules for the guidance of the accoucheur. Solomon Naumann[15] (1762) and Murray[16] (1797) wrote elaborate treatises on the subject, whilst Camper carefully studied its pathology in 1785, and Baudelocque[17] gave valuable indications as to diagnosis, prognosis and treatment. Since that time various observers, including Simpson,[18] Cazeaux,[19] Tarnier,[20] Depaul[21] and especially Chassinat[22] have discussed the subject in extensive papers. Lately, the most important works have been those of Ramsbotham[23] in 1867, McDonald[24] in 1878, Fritsch[25] in 1876 and Klebs[26] in the same year. In most text-books on midwifery, especially those of Spiegelberg, Schröder, Lusk, and Charpentier, a good account is given of the disease in its relations to midwifery.

The last paper, that of Poullet,[27] written in 1880, is perhaps the best sketch of the disease in its relations to midwifery that has been written up to the present time.

But although the disease has been studied by many observers, the pathology of this affection is still enveloped in many obscurities. Tulpius,[28] Wepfer,[29] Bonnet,[30] Valsalva,[31] Morgagni,[32] Lieutaud,[33] give *post-mortem* records of hydrocephalic patients, but the mode of increase in the size of the skull and the ætiology of the disease are hardly mentioned. J. L. Petit, in 1718, Robert Whytt,[34] as well as John Warren[35] in 1788, added to the knowledge already gained and proved the existence of a chronic form of the disease in contradiction to Fothergill's[36] opinion, who believed in the existence of the acute form only. Morgagni had, however, before them fully discussed the pathology of the disease. These authors agree in finding the dura mater more adherent than usual, the base of the brain flattened and the ventricles so much distended that the grey and white substance of the brain measure but a few lines in thickness.

More lately, especially since the discovery of the cerebro-

spinal fluid by Magendie and of the foramen bearing his name, the pathological appearances have been assumed by some to be the result of an obstruction to the free flow of the cerebro-spinal fluid. Hilton[37] seems to have been the first to propagate this idea. It was then taken up by Archambault and others, and mentioned in all the text-books of pathological anatomy. Although an obstruction undoubtedly exists in many cases, it is, in the writer's opinion, frequently absent, or, on the other hand, may be present without occasioning hydrocephalus.

During the present century the disease has found a place in all text-books of pathology (Virchow, Lancereaux, Cornil et Ranvier, Ziegler, &c.), but little that is new has been discovered. Kundrat's[38] paper is a most valuable contribution to the study of one form of the disease, but most records of cases and *post-mortem* examinations are found scattered in the transactions of many medical societies.

In this paper the writer hopes to have made use of nearly all the literature of the subject, and to give a fairly full account of what is known of this affection.

I.

The brain in the embryo[39] presents itself in the anterior dilated portion of the primitive medullary tube, and forms the three primary cerebral vesicles. These primary cerebral vesicles are named fore-brain, mid-brain, and hind-brain, and correspond most nearly to the regions of the *third ventricle*, the *corpora quadrigemina* and the *medulla oblongata* of the adult brain. The changes which mainly tend to modify the form of the primitive brain are:—

1st. The development on each side from the anterior vesicle of the primitive ocular vesicle.

2nd. The protrusion somewhat later from the fore part of the anterior cerebral vesicle of a bulging part, at first single or undivided, but which, by a median cleft and lateral expansion, becomes later the rudiment of the true cerebral hemispheres.

3rd. The formation in the fore part of the posterior vesicle of a new encephalic rudiment corresponding to a cerebellum. Thus, the first vesicle becomes converted into

the cerebral hemispheres and vesicle of the third ventricle or thalamencephalon; the middle vesicle remains undivided and the hinder vesicle becomes the cerebellum and medulla oblongata.

There are thus distinguished the rudiments of five fundamental constituents of the adult brain.

1st. The cerebral hemispheres with their ventricular hollows or lateral ventricles, the *corpora striata* and the olfactory lobes—a set of parts to which, as a whole, the name of pro-cerebrum or pro-encephalon may be given.

2nd. The thalamencephalon with its cavity or third ventricle, the primary ocular pedicles and the infundibulum.

3rd. The mesencephalon comprising the corpora quadrigemina and crura cerebri with its contracted internal hollow—the *iter a tertio ad quartum ventriculum* of human anatomy.

4th. The next part in succession is the cerebellum, along with which is included the pons Varolii and part of the fourth ventricle.

5th. The hinder part, which passes into the spinal marrow, is the *medulla oblongata*, with the remainder of the fourth ventricle and its continuation into the central spinal canal.

In the adult the brain contains five cavities, which are all in communication, namely: (1) the two lateral ventricles, communicating by the foramen of Monro with the (2) third ventricle, communicating through the iter a tertio with the (3) fourth ventricle, which itself communicates with the spinal canal by the foramen of Magendie.

Further, the ventricles of the brain communicate with the sub-arachnoid space by means of the foramen of Magendie, the opening into the lower part of the fourth ventricle, through the expansion of the pia mater (tela choroidea inferior) which closes it. Two other openings through the membrane exist, one on each side behind the upper roots of the glosso-pharyngeal nerve, in the pouch-like extension of the membrane beneath the flocculus.

A certain quantity of the fluid is contained between the arachnoid membrane and the dura-mater; but the larger

quantity of the cerebro-spinal fluid is lodged in the sub-arachnoid space in the meshes of the trabecular tissue. It must be remembered that fluid injected into the sub-arachnoid space passes freely into the Pacchionian bodies, is found after a time to filter through their walls and thus to get into the sub-dural space, although there does not appear to be any open communication between the interior of these bodies and the prolongation of the subdural space which surrounds them. Moreover, if the injection be continued, it can be driven even into the interior of the venous sinuses and lacunæ which are found in connection with them, especially into the superior longitudinal sinus, into which the arachnoidal villi project. Thus these villi seem to afford a means of passage of the cerebro-spinal fluid from the sub-arachnoid space into the venous sinuses, when the fluid pressure in the sub-arachnoid space becomes from any cause increased above the normal.

Lastly, Axel Key and Retzius have been able to inject the ventricles of the brain by introducing finely-divided coloured fluids into the sub-arachnoid space.

Hence collateral channels are provided by nature for the cerebro-spinal fluid to escape in case of closure of Magendie's foramen. Whether the collateral circulation in case of closure of the latter foramen ever becomes quite adequate to the extra work, remains as yet an open question.

Whilst reading through the literature of the subject the writer met with the records of a number of cases in which only part of the brain is converted into a kind of cyst, this pathological cavity often, though not always, communicating with the ventricles (*porocephalus*).

It was his intention at first to exclude all such cases, but on further consideration he found it impossible to do so, as, for reasons to be stated later on, he came to the conclusion that there was no rigid line of demarcation between *porocephalus* and *chronic hydrocephalus*. In this paper, therefore, the two conditions will be discussed together, although for the sake of clearness the pathological anatomy and clinical symptoms of each will be spoken of separately.

The localisations of each disease are first described; secondly, the morbid changes in the walls of the cavity containing the fluid and in the vessels of that part; and, lastly, the changes in the skull and other parts of the body will be studied.

In the medical literature of this century the records of *post-mortem* examinations in which *localised* collections of fluid in the brain have been found are but few, and the writer, after a careful search, has been unable to collect more than 114 cases of this rare affection.

In 30 per cent. of these both sides of the brain were almost completely destroyed by the contained fluid, and in 10 per cent. the whole of one hemisphere was similarly affected. No marked disproportion existed between the number of cases in which the one or the other hemisphere was the seat of disease. In 52 per cent. the lesion was limited to part of one hemisphere.

In 15 per cent. of these the anterior portion of the brain was the seat of the disease, in 61 per cent. the middle part, and in 16 per cent. the posterior lobes were affected. In 5 per cent. the lesion was situated in both the anterior and middle part, and in 1 per cent. in the cerebellum.

Kundrat's results differ slightly from these. This author states that in twenty-seven cases the seat of the disease was found in the parts supplied by the middle cerebral arteries, in three in the region of the anterior cerebral and in five cases in that of the posterior cerebral.

In 8·7 per cent. of cases tabulated by the writer, one horn of one ventricle only was dilated, the dilatation being most frequently present in the lateral and in the fourth ventricles (no cases of meningocele or encephalocele are included in these statistics). Moreover, a few cases have been excluded in which the lesions were very small and bilateral, or recorded with insufficient minuteness to admit of classification.

The cavity containing fluid communicates with the lateral ventricles in a large number of patients, and this is the case not only when both or the whole of one hemisphere having been destroyed, the brain or one side of it is con-

verted into one huge cavity containing fluid, but also in the slighter forms of destruction.

In 46 per cent. it is distinctly noted that the fluid passed with the greatest ease from the cyst into the lateral ventricles, or *vice versâ*. In the other accounts of *post-mortem* examinations no mention whatsoever is made as to the presence of a communication between the cyst and the normal cavities existing in the brain. The following cases have been selected among many others as typical examples of some of the pathological appearances found in these patients.

In a child[40] born at full term, the surface of the cranium was covered by a red membrane, stretched over a mere crust of bone. On removing this crust some serum escaped. Both cerebral hemispheres and the cerebellum were absent. In another patient[41] the roof of the skull was lined by a vascular membrane mottled with dark red spots like an old apoplectic cavity, another very dense and brownish-red membrane covering the base of the brain. The base itself consisted of the following structures:

(*a*) Corpora striata, brownish red in colour, indurated and unequal in size.

(*b*) Optic thalamus.

(*c*) Some atrophied convolutions at the base of the brain. The olfactory nerves were atrophied, and only traces of the optic commissures and anterior pyramids were found, but the olivary bodies, tubercula quadrigemina, pons and cerebellar peduncles were healthy. Nothing remained of the convexity of the brain, in another case, but a thin greyish layer applied to the falx cerebri and a few traces of convolutions at the base. Cruveilhier[42] has also described another specimen, in which the cortex of the cerebral hemispheres was replaced by a thin brownish membrane covered with granulations. The convolutions were represented by a few very small and dense little masses, forming irregular cylinders curved on themselves. Other noticeable points were the complete absence of the pyramids and large development of the olivary bodies. The cerebellum and cerebellar peduncles were quite healthy, whilst the optic nerves were atrophied and the olfactory nerves absent.

Schwarz[43] could not find a trace of cerebral hemispheres in a patient whose pons, medulla and cerebellum remained intact, the superior sympathetic ganglion being slightly hypertrophied.

The pons, medulla, cerebral peduncles, corpora striata, and optic thalami were quite normal in a specimen shown by Budin[44] to the Société Anatomique of Paris, but the two cerebral hemispheres were represented by the temporal and occipital lobes only, which extended to the internal occipital fissure. Duret,[45] who examined this brain, noticed that the distribution of the lesion corresponded to the areas supplied by the anterior cerebral and sylvian arteries. The carotids were small, and the sylvian and anterior cerebral arteries resembled those of a fœtus. He thought, therefore, that a disease of the membranes (congestion, hæmorrhage) at some period of fœtal life (four months) had been the cause of the malformation of the hemispheres. A fact in favour of this view was that the internal aspect of the pia mater over the anterior lobes had a rusty tint like that found in the site of an old hæmorrhage. Dickson[46] recorded a very similar case in 1838.

In a patient whose case has been reported by Binswanger[47] there was a complete atrophy of the third left frontal convolution, of the two inferior thirds of the ascending parietal and ascending frontal convolutions; the angular gyrus, first temporal and lobule of the insula being almost wholly destroyed. The atrophy was therefore almost limited to the parts supplied by the sylvian artery, this vessel not being discoverable. The ventricle on the same side was dilated, but the right hemisphere had not undergone any compensatory enlargement. In a girl, twenty-six years old, Frigario[48] found at the level of the right parietal lobe a sac containing about eighty grammes of yellow and transparent liquid, the smooth walls of the cyst being continuous with the dura mater. The choroid plexus lay at the bottom of the sac, together with one extremity of the right optic thalamus. The cerebral lesions affected the right motor region, the ascending parietal, superior and inferior parietal lobules, part of the angular gyrus and superior part of the quadrate lobe.

On the left side the circumvolutions of the quadrate lobe were rudimentary. An interesting case published by Dr. Cunningham[49] in the thirteenth volume of the *Journal of Anatomy* (p. 508) may also be included in this category.

The walls of the cyst have not always been carefully described. Schwartz,[50] however, mentions that the membranes covering the cavity were formed by an outer layer—the dura mater—and an inner very thin and fragile layer, which appeared to be the ependyma.

According to Kundrat,[51] the meninges are usually healthy. Cotard[52] describes the meninges as being bound more or less extensively to the ventricular membrane. Vessels run between these two membranes and near the boundaries of the cavity, the wrinkled convolutions being transformed into a gelatinous substance before they wholly disappear.

The lateral boundaries are, in the majority of cases, perfectly smooth, but columns and ridges running along the walls of the cyst have been described.

The ventricle is sometimes, though rarely, dilated as a whole; more frequently one horn only is the seat of disease and is more or less cut off by adhesions from the remainder of the cavity.

G. Vrolik[53] has described a specimen in which the anterior horn of one lateral ventricle was cut off as the result of a former inflammatory process. Dropsy taking place in the diverticulum thus isolated, the brain and corresponding part of the skull on the same side were greatly distended. Virchow[54] states that the posterior horn of one lateral ventricle may be obliterated either in its entire length or in part only, forming (encysted hydrops of the posterior horn) a cavity which sometimes does not communicate at all with the ventricle and may then become the seat of morbid processes. The fourth ventricle is itself sometimes the seat of a limited effusion, and the same author has figured a localised and encysted dilatation of the fourth ventricle pressing on the facial nerve and producing a paralysis of the same. Axel Key and Retzius mention this case in their classical work, and suggest that this pathological condition was possibly due to the closure of the aperturæ laterales ventriculi quarti on the same side.

Hamel has seen unilateral vaso-motor paralysis of the face associated with congenital hydrops of the fourth ventricle. More lately Hemming,[55] writing in the *British Medical Journal* for 1887, reports a valuable case in which, at the *post-mortem*, he found the fourth ventricle distended by a cyst which occupied the whole cavity, had burrowed into the cerebellum and was filled with yellow serous fluid.

Hydrops of septum lucidum and pituitary body, with or without other effusions in the brain, have also been noted.

All these appearances must be, of course, distinguished from the cysts formed at the seats of an old hæmorrhage, &c.

In many cases of localised effusions in the horns of one of the ventricles, the dilatation gives rise to a hydro-encephalocele. Some authors go so far as to say that the liquid in a hydro-encephalocele is always due to dropsy taking place in a limited part of the cerebral ventricles. It is not intended to give here a full account of this variety of disease, but merely to show the relation existing between it and hydrocephalus.

In the vast majority of patients, the protrusion takes place in the occipital region, generally above the occiput, more rarely below it, or on the sides. In no less than 135 out of the 220 cases collected in Lawrence's,[56] Springs, and Larger's[57] papers, the hernia protruded in the occipital region. An important point is that the tumour does not pass through a fontanelle, as is sometimes described, but always through a bone. The frontal region, at the junction of the frontal and nasal bones, is another favourite place for its appearance; occasionally the protrusion takes place through the frontal bone itself.

Houel[58] states that the hernia may pass out through the following apertures: the os unguis at the level of the inner angle of the orbit, the sphenoid fissure, the body of the sphenoid or inner portion of its wings, the cribriform plate of the ethmoid and the sphenoidal sinus. Stein[59] has seen the tumour pass through the sphenoid fissure, follow the cavernous sinus on the right side, pass through the nose, and end in the mouth.

The centre of the tumour consists of part of the nervous

centres, the protruding part of course varying in size, the whole being covered by the red, thickened and villous pia mater. If the opening be small there is a distinct neck to the tumour; if large the cerebral hernia takes place *en masse*. The liquid accumulated in the ventricles pushes the cerebral matter before it, the convolutions gradually unfold, become thinner, adhere to the dura-mater and may even disappear. If the cerebral matter gives way, the herniated part collapses, forms a shrivelled stump, whilst the liquid is contained in a cyst on the exterior and may, through a fistula, communicate with the fluid inside. The ependyma, as a rule, shows signs of chronic inflammation. The same deformities of the body so frequently met with in cases of chronic hydrocephalus are often present in this variety. In only very few cases is there any mention made as to the state of the central canal of the cord, of the aqueduct of Sylvius, or as to evidences of obstruction in the central canal. In one patient the base of the brain is said to have been covered by a thick membrane, and in another there was a dilatation of the aqueduct of Sylvius, the foramen of Magendie being closed by a fibrous membrane.[60]

True chronic hydrocephalus differs from porocephalus only in the fact that the disease affects both ventricles without there being an actual loss of continuity in the cortical substance. The fluid when occupying both lateral ventricles often distends the latter to an enormous extent, these cavities occasionally containing as much as twenty-five pints of fluid. The ventricles lose their shape almost completely and, through destruction of intervening tissues, become converted into one huge cavity, the middle one being absorbed into the other two. The distension, however, is not uniform and is always more marked on one side or even in one horn of a ventricle than on the other side. In one brain examined by the writer, not only was the left side of the skull and brain distinctly larger than the right, but, on examining the left lateral ventricle, the middle part and anterior cornua were found to have been far more distended by the fluid than the posterior.

The corpus callosum is often displaced and forced upwards so as to lie on a level with the upper surface of the hemispheres. The nervous substance entering into its formation sometimes almost completely disappears and is then represented by a transparent[61] fibrous membrane only, which is thrown transversely across and presents in the middle line a kind of fold in which some fibres of the cerebral substance still exist. In one case this membrane was formed by a fibro-vascular layer continuous with the pia mater and a solid membrane lining the corresponding lateral ventricle.

The foramina of Monro, when both lateral ventricles are the seat of disease, are enlarged and measure sometimes as much as 3½ inches across. When the distension is unilateral the foramen on the same side has been found closed.[62]

The aqueduct of Sylvius is not wider in all cases—in fact its channel as well as the fourth ventricle are for the most part quite normal. The lining membrane of the ventricle shows well-marked pathological changes. It is often thickened and of a leathery consistence and either strips off too easily[63] or else is unduly adherent. It is rough, covered with small bands or columns running from side to side or with fibrous tracts and false membranes extending across the ventricles. These fibrous bands running along the walls of the ventricles are not unlike the columnæ carneæ of the heart. The surface of the membrane is shaggy, covered with small granulations or with small sago-like transparent bodies, the nature of which remains uncertain.

The most important point in the pathological anatomy of the disease, however, is the obstruction to the passage of fluid from one ventricle to the other, of which this thickened membrane may be the cause.

In a typical case described by Archambault,[64] a thick and resistant membrane formed the internal part of the walls of the ventricular cavities, numerous vessels and the cerebral substratum being visible below. This was simply the membrane normally covering the ventricles, but, being thickened, it was not unlike that found in the interior of a cyst. It lined the ventricular cavities, penetrated into the middle cavity through Monro's foramen and some pathological apertures,

passed over the orifice of the aqueduct of Sylvius into the third ventricle without dipping into it and thus closed the cavity.

This case illustrates the lesion most frequently found in hydrocephalus, namely, obstruction in some point of the canal running through the middle of the cerebro-spinal axis.

The obstruction may be lower down in cases where at the *post-mortem* the aqueduct and fourth ventricle are distended. In a specimen shown by Dr. Baxter[65] to the Pathological Society the cerebro-spinal opening was distinctly obliterated; for, on passing a probe backwards through the dilated iter a tertio, its point was felt to be stopped just below the calamus scriptorius by a thin but opaque membrane, the result of some former attack of very circumscribed meningitis. Magendie[66] himself has collected eight or ten observations of chronic hydrocephalus in which he had found the foramen first described by him closed by inflammatory adhesions, and Hilton,[67] in his lectures on rest and pain, mentions the same lesion as a cause of hydrocephalus.

Cruveilhier[68] states that Magendie's foramen was closed by a fibrous membrane in one case, and the same condition must have existed in Luton's[69] specimen. An obstruction, however, is not always present. It was distinctly absent, for instance, in the cases recorded by Mr. Hutchinson[70] and Gaucher.[71] In the specimen described by Mr. Hutchinson for instance, the cavity of a spina bifida was in direct communication with the brain by means of the enlarged central canal.

The choroid plexuses are often hypertrophied and their vessels diseased. In one patient the plexus choroideus medius running from the foramen of Monro to the posterior cornu was very thin, but the plexus here became as thick as a walnut, diminishing in size further on, the vessel walls being healthy. The plexus are occasionally bound to the adjoining tissues by inflammatory adhesions so strongly that their removal becomes impossible without inflicting injury on the neighbouring structures.

Cystic alterations in the connective tissue cells of the vessels' walls have also been described, and meningeal and other hæmorrhages in the cranial cavity are of frequent occurrence. The vessels likewise are often enlarged.

In a very interesting *post-mortem* examination of a hydrocephalic patient made by Dr. Dickinson,[72] an extra ventricle was formed by the tentorium and the upper surface of the cerebellum. This cavity was in free communication with each lateral ventricle by a large rounded opening on the outer surface of the transverse fissure on either side. The fourth ventricle and the passage leading to the third were perfectly normal, though there was some thickening of the arachnoid membrane between the cerebellum and spine, as also about the base of the brain.

The distension of the ventricles of the brain of a patient suffering from chronic hydrocephalus (age 50) which the writer had an opportunity of examining was more marked in the middle and in the anterior regions; consequently, the pathological changes in the convolutions were more evident anteriorly also. The cerebral substance over the frontal and orbital lobes was not more than one centimetre thick, whilst behind the Rolandic fissure the amount of brain-tissue gradually increased, so that, posteriorly, it amounted to double that in front.

The paracentral lobules were flattened, while the corpora striata and optic thalami appeared to be somewhat compressed, though otherwise normal. On examining the exterior of the brain the convolutions were still distinct; the sulci, however, much shallower than those of a normal brain. All the fissures were visible still, the sulci and convolutions in the occipital region being almost normal in appearance.

Distinct flattening of the ascending parietal and all the frontal convolutions, as well as of the paracentral lobule and the anterior part of the callosal and calloso-marginal convolutions, was noticeable; whilst the orbital part of the brain was almost normal in appearance. The island of Reil, however, had been forced outwards by the cerebro-spinal fluid and its surface lay level with the neighbouring convolutions, the whole of the insula being visible without any dissection. An obstruction to the flow of cerebro-spinal fluid could not be found anywhere.

The microscopic examination of this brain was made after hardening the organ in a weak solution of chromic acid, the pons, cerebellum and each hemisphere being treated separately. After hardening, small pieces were removed from the ascending first, second and third frontal convolutions, island of Reil, orbital, occipi-

tal, parietal and temporo-sphenoidal convolutions, also out of the angular gyrus, paracentral lobule, and sections made of these parts. The pons medulla, internal capsule and corpora striata were examined separately. The sections were stained in an ammoniacal solution of carmine, this fluid having been found to give the best results.

No lesions were found in the internal capsule or corpora striata and no tracts of degeneration in the pons or medulla. The only change found in the pons was that the central canal was almost, though not completely, filled with epithelial *débris* and that its margins presented an irregularly festooned border. The grey matter around it was softer than natural, breaking down easily and staining somewhat more deeply than the grey matter elsewhere, but no distinct histological changes could be discovered.

The cortical substance of the brain, however, showed well-marked pathological appearances, varying in character according to the different regions of the brain.

On holding to the light a section of the middle part of the ascending frontal, the grey matter, as is usually the case, appears to be stained more deeply than the white substance. Further, comparing this section with one from a normal brain, the layer of grey substance in the former is distinctly thinner by about one-sixth. On more careful examination, the grey matter is divided into three strata:—

1. A very small superficial stratum corresponding to the first cortical layer, stained somewhat less than the other parts of the grey matter.

2. A very thin stratum immediately underneath the first. This part contrasts sharply both with the stratum above and below, in staining far more deeply with carmine than the parts surrounding it.

3. The remainder of the grey matter which stains more deeply than the first, but far less than the second stratum.

On microscopic examination the first stratum consists of the first superficial layer of grey matter. This is perfectly healthy and normal in size and appearance. The second or deeply stained stratum is limited to the second layer of grey matter and contrasts very sharply with the parts above and below it. The deep colouration is due to a great excess of neuroglia, and when examined with a high power this layer is found to consist almost wholly of a very dense reticulum of fine connective tissue con-

taining a great many nuclei. The nerve-cells have almost completely disappeared in that layer, but the few which remain, judging from their size and shape, appear to be healthy.

The axis-cylinders have completely disappeared in some places. In other parts they are far less numerous than in a normal brain. This lesion is strictly limited to the second layer, the grey matter below, if somewhat reduced in amount, being otherwise quite healthy. The white matter is somewhat more vascular than usual, but no morbid changes of any kind are to be detected. The same lesion is also noticeable in the parietal convolutions, in the angular gyrus, in the island of Reil and in the paracentral lobule, as also in the callosal, calloso-marginal and marginal convolutions. In the island of Reil and paracentral lobule, however, the sclerosis though still limited to this second layer, is far more marked, the nerve-cells and axis-cylinders in the second stratum having completely disappeared in the grey matter covering the island of Reil.

In the occipital region the lesions are somewhat different. The first or superficial layer stains far more deeply with carmine than the regions previously described and microscopic examination reveals a considerable excess of connective tissue in this part. The second layer is quite healthy, but the third or intermediate stratum like the first shows a well-marked hyperplasia of connective tissue. The hyperplasia is not limited to this stratum, for in some places small microscopical bands of hyperplastic connective tissue extend from this layer into the deeper parts of the grey matter, where also the connective tissue is in excess.

The white matter below contains a considerable number of blood-vessels and shows an increase in the connective tissue. The nerve-cells are somewhat fewer in number in the places where the connective tissue is in excess, but the nervous elements do not seem to have suffered much, the axis-cylinders having a normal appearance and the nerve-cells, if somewhat less numerous, quite healthy.

No changes whatsoever can be detected in the sphenoparietal lobes, but in the orbital lobes distinct pathological alterations are found. Scattered through the first layer are a number of nucleated star-shaped cells, which are not nerve-cells, but connective-tissue elements forming little islands of sclerosis lying in the middle of healthy tissue. The nerve-cells in the deeper layers are surrounded by a large clear space and the cells and nuclei are filled with very fine granules—a lesion not unlike that which has been described as occurring in senile dementia. In the deeper parts these appearances are more marked still, the cells being

very granular and the spaces around them larger. But, strangely enough, there is no excess of connective tissue in this part of the brain. The optic nerves were examined, but no changes could be detected. To resume: bands of sclerosis were found extending over various parts of the brain. These bands affected one or several layers, according to the part of the brain affected, not spreading, as a rule, to the other layers. The nerve cells were not markedly affected except in one part, where they showed the lesions which are characteristic of senile dementia.

The interest of these lesions consists in the fact that a morbid process affecting the whole of the brain produces pathological changes varying in extent according to the part of the brain examined. Thus in the motor region the lesion is limited to the second, in the occipital region to three other layers. These changes resemble somewhat those found in the spinal cord where degenerations are often limited to certain definite tracts (motor sensory). It will be interesting to see, therefore, whether or not there is some corrèlation in the development or functions of some of these layers. Lastly, the lesions demonstrate the changes produced by long-continued pressure acting on the grey substance of the hemispheres.

The convolutions are often far more flattened than in the brain just described, the sulci becoming completely obliterated even, the whole brain being converted into one large membranous bag a few millimetres thick. Part of the brain or the membranes covering it may be forced out through a normal (fontanelle?) or pathological opening—through a bone even—and give rise to a true hernia cerebri.

The base of the brain, as a rule, is far less affected than the vault, or may be quite normal. The corpora striata, optic thalami, and other ganglia at the base are flattened and lengthened, or one corpus striatum and optic thalamus presents a normal appearance, whilst the other may have almost disappeared—another proof of the often asymmetrical distension of the head.

When the aqueduct of Sylvius is enlarged, the pons and cerebral peduncles are also flattened and altered in shape.

Virchow[73] has drawn attention to the presence of nodo-

sities of cerebral matter in cases of congenital hydrocephalus, these nodosities forming a kind of excrescence on the surface of the cerebral ventricles. They are especially met with in places where, in healthy brains, the surface of the ventricle is quite smooth—namely, just at the margins of the corpora striata and optic thalami. This condition, according to the same author, may be partly due to irritation. The membranes covering the brain are often somewhat thickened and occasionally adherent, presenting signs of recent meningitis,[74] but, as a rule, the lesions are by no means characteristic. Small pedunculated cysts hanging into the ventricles are occasionally present but of little importance.[75]

The cranium always presents characteristic lesions. In congenital porocephalus the head is normal in size, or even smaller than normal. Cruveilhier[76] has shown that premature ossification often takes place, the bones being frequently very thick and the sutures uniting too early. In chronic hydrocephalus the head may vary from just above normal to an immense size. The largest on record is a fœtal head which was in Meckel's[77] possession, the biparietal diameter measuring forty-three centimetres, and another described by Frank, which measured 140 centimetres in circumference. These enormous dimensions are very rare, however. Vinsoneau[78] who has minutely examined this question, finds that the average hydrocephalic head exceeds the normal (1845 ccs.) by 244 ccs., and it is useless to enter into further details as to size.

A hydrocephalic cranium is always asymmetrical and measurement shows that one lateral half of the head is always larger than the other. The writer has accurately measured fourteen dried crania of hydrocephalic children and adults, and has found that measurement from the occipital protuberance to the root of the nose is always larger by three-quarter centimetres at least on one side than on the other, the difference on one occasion amounting to two and a-half centimetres, but averaging one and a-quarter centimetres.

Not only may one side be the larger, but the anterior part

may be increased out of all proportion to the posterior half, or *vice versâ*. Moreover, one very limited part of it, one or more bones, one frontal or occipital for instance, is sometimes expanded out of all proportion.[79]

The lateral distension is, as a rule, more marked than the antero-posterior. Thus Vinsoneau[80] found that out of thirty-two heads examined by him eighteen were brachy-cephalic, ten subbrachy-cephalic, and four dolicho-cephalic. The hydrocephalic crania may be divided into the following classes:—

Class I.—General enlargement.

Class II.—Partial enlargement.

(*a*) Anterior hydrocephalus, where the frontals are pushed forwards and irregularly ossified. (*Tête à bonnet à poils. Giraldès*).

(*b*) Posterior hydrocephalus.

(*c*) Cases where the parietal bones being prematurely ossified, and the antero-posterior diameter being lengthened, the lateral parts chiefly give way (scapho-cephalus). Out of thirty-seven observations in Vinsoneau's paper only two showed this character.

(*d*) Cases where, owing to the partial enlargement of one ventricle, a few bones or only one are deformed.

The bones forming the vault of the skull are thin, as a rule, and transmit light readily, but in certain places they are sometimes thicker than natural. If the convolutions were still well marked at the time of death, their impressions, even to their minutest sulci, are sometimes marked on the bony skull. This was typically the case in one cranium which the writer, thanks to Professor Pierret's kindness, had an opportunity of examining at Bron Asylum.

On holding up the skulls to the light, places where the ossification is deficient are frequently seen. These points of deficient ossification, scattered more especially on the frontal and temporal bones are often merely covered by a lamina of bone, if not actually perforated. Large holes are often present in some of the bones, chiefly the frontals, through which a hernia cerebri sometimes protrudes.

The bones of the adult skull are often quite as thick,

or even much thicker than natural, though points where the ossification is deficient are often noticeable. The anterior fontanelle is sometimes covered by a thin layer of bone only, or the inter-frontal and other sutures are visible long after the time at which they usually disappear, showing that, in spite of the often enormous thickness of the skull, some further distension was still taking place at the time of death. The frontal, parietal, squamous portion of the temporal and occipital bones are those which suffer most. The frontal bones pushed forwards are often almost twice as large as natural, retarded in their union, or one or both of them are cleft through the middle. The enlarged parietals often differ in size and present the lacunæ above mentioned. In one cranium which the writer had an opportunity of examining the left parietal bone had evidently been divided into two, and traces of an accessory suture running from before backwards still remained at the time of death, which occurred at the age of fifty-four.

The occipital bone is almost always distinctly asymmetrical and, in children, a fissure often runs from its point of junction with the parietal bones to the occipital foramen. It is occasionally driven downwards and backwards so as to become almost horizontal. The foramen magnum is altered in shape, asymmetrical and oblique, on account of frequent malformation of the occipital bone.

Ossification of the sutures is occasionally premature, at other times retarded, both conditions existing in the same skull sometimes. Thus the writer has seen a dried skull of a child aged two and a-half years, in which the anterior fontanelle being still open and the lambdoid and coronal sutures still half a centimetre apart, the inter-frontal suture, which as a rule is the last to close in hydrocephalic children, was firmly ossified.

The sutures are sometimes linear, their margins being thin, almost straight and not dentated, a small fibrous band only showing between the bones. Occasionally they measure several millimetres or even centimetres in width. A chain of Wormian bones often forms in a suture[81] and keeps the bones apart. In other specimens the sutures appear to

be intimately joined, but nevertheless the bones can be disarticulated with the greatest ease.

Atypical sutures have also been described. The anterior fontanelle[82] often remains patent for a long time and its situation is often visible, even in the skulls of patients who died at an adult age. Cazalis mentions the case of a child $9\frac{1}{2}$ months old whose anterior fontanelle measured $4\frac{1}{2}$ centimetres across. In a girl sixteen years old Fürst[83] found the anterior fontanelle patent. The fontanelle of Gerdy and other accessory fontanelles are sometimes present, though the posterior and lateral fontanelles are generally ossified early.

Wormian bones are small accessory bones often present in the crania of hydrocephalic patients. They are small, generally placed on the sutures, often symmetrical and more frequently found on the posterior surface of the skull than on the anterior.[84] The lambdoid suture is their favourite situation, though according to some, they are most often present on the coronal and sagittal sutures. The face is almost always free of them. Vinsoneau,[85] however, describes one skull in which a Wormian bone was placed on the naso-frontal suture. There may be as many as ninety on one skull, but sometimes only one or two; their size, according to Sappey,[86] varying in an indirect ratio to their number. One Wormian bone placed exactly over the posterior fontanelle and articulating with the occipital and parietal bones, is probably only the highest part of the occipital which has remained distinct. It is a small lozenge-shaped or triangular bone often divided by fissures into two or three distinct portions (os epactale[87]).

Sir Prescott Hewett[88] describes a cranium in which the fore parts of the squamous portions of the temporals, as well as the great wings of the sphenoids, driven downwards and outwards, bulge into the zygomatic fossæ and lie on a level with the margins of the superior maxillary bones. The glenoid cavities in another skull are six inches, the two external angular processes of the frontal bones $7\frac{3}{4}$ in. apart. In one skull there is on each side a large pouch bulging outwards and projecting forwards into the region of the cheek,

as if the child's cheeks were blown out. The meatus auditorius externus is displaced to the under surface of the skull, below the level of the foramen magnum, and almost in contact with the condyloid process of the occipital. The anterior and middle fossæ of the skull are larger than natural, and defects in the ossification of the base of the skull, in the shape of premature or deficient ossification, are noticeable points in many skulls.

The orbital plates, especially in young children, are driven downwards and either present a plane surface oblique from before backwards, or are perpendicular, even convex, bulging into the orbit and sometimes reducing the latter to a mere chink. The orbital arch is more or less obliterated, the frontal and orbital portions of the bone sometimes forming one continuous line convex in its whole length. The orbits of adults, owing probably to the lesser degree of distension, are sometimes almost normal.

Though the face in these cases looks small on account of the enormous distension of the skull, it is quite as large if not larger than usual. The writer has measured the distances between the two malar bones and from the root of the nose to the chin in fourteen hydrocephalic skulls, and has found them to be quite as large as in normal children. In fact the distance between the two malar bones is as a rule increased by about 0c.5 in the hydrocephalic children measured by the writer. The face loses its oval shape and assumes that of a triangle with its apex at the chin. The root of the nose is often somewhat broad and the lower jaw prominent. In all cases examined by the writer the palate was somewhat narrow and high arched, the teeth were irregular, grooved longitudinally and often carious. The development of the teeth is not only retarded but premature caries is the rule. Bouchut[89] states that he has seen well-formed teeth in hydrocephalic children. This may be the case occasionally, but almost all the writers on the subject speak of the teeth as being retarded in their development and irregular in shape. In the Musée Dupuytrin in Paris there are more than twenty hydrocephalic crania. In many of them the teeth are completely missing, the alveoli having also disappeared, whilst in

the few in which the teeth are present they are invariably carious.

Many authors have noticed the coincidence of hydrocephalus with spina bifida, but the writer is unable to give any statistics as to the frequency of the coincidence. It has been reported that after the cure of spina bifida the child had become hydrocephalic, and it has been argued that the hydrocephalus in these cases was due to the cure of the spina bifida. It might, however, be argued with far more probability that the cases in which the symptoms were first noticed after the cure of spina bifida were cases in which the symptoms of hydrocephalus began some considerable time after birth. Dr. Barnes's[90] case is very interesting from that point of view, for though the spina bifida was congenital the hydrocephalus did not make its appearance till the patient was nine months old.

Analyses of Cerebro-spinal Fluid in Cases of Hydrocephalus.

TIDY.[91]		YVON.[92]	
Water	1000 parts	Sp. gr....	1008
Solids	5.18 parts		
Albumen	·98	Organic matter...	6·100
Sugar	·63	Mineral matters	9·1
Carbonate of soda	·02	Fixed substances	15·1
Sulphate of soda	·08	Albumen	·900
Chloride of sodium	2·14	Total albumen	2·040
Chloride of potassium... ...	·6	Urea	2·800
Fat	·1	Fatty matters	·120
Alcohol extract...	·63	Phosphoric acid	1·291
Total	5·18	Sulphuric acid	·117
		Calcium	·089
		Chloride of sodium	7·
		Water	994·900
		Total	1000

HILGER.[23]		PAPP.[24]	
Water...	98·775	Sp. gr.	1007·078
Solids...	1·225		
Salts	·762	Chloride of sodium	·32%
Albumen	·246%	Carbonate of soda	·41%
Sugar	·164%	Phosphate of soda	·02%
		Phosphate of calcium... ...	·01%
			·76%
Chloride of sodium	·397	or	
Chloride of potassium	·682	Soluble salts	·706
Sulphate of potassium	·032	Alcoholic extractives	·004
Phosphate of potash	·124	Albumen	·181
Phosphate calcium, Magnesium, Iron	·096	Water	99·049
Total	100	Total	100

Other deformities sometimes associated with hydrocephalus are hare-lip, cleft palate, absence of thoracic and abdominal organs, transposition of organs, talipes, pes equinus, equino-valgus or varus, double fingers, malformation of arms, legs and feet, or absence of one or more bones.

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